ARCHIVES OF OPHTHALMOLOGY

VOLUME 18 (old series Volume 75) DECEMBER 1937 NUMBER 6

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CHIASMAL SYNDROMES PRODUCED BY LESIONS IN THE POSTERIOR FOSSA

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The symptoms of lesions producing pressure on the optic chiasm are extremely variable. While various bitemporal hemianopic defects are the most frequent and most characteristic type of changes found in the perimetric fields, asymmetrical or incongruous altitudinal anopsias and homonymous hemianopias are frequently encountered. Unilateral or bilateral complete amaurosis is ascribed usually to a lesion of the optic nerve proper, and in the absence of an ophthalmoscopically visible explanation for its presence it is usually interpreted as evidence of a lesion in the chiasm or in the optic nerve anterior to the chiasm.

In association with choked disks, defects in the perimetric fields suggesting pressure on the prechiasmal portions of the optic nerves or on the optic chiasm itself are interpreted usually as evidence of basofrontal tumor, suprasellar tumor of the Rathke pouch type or tumor of the third ventricle. Ordinarily these diagnoses will be correct. Occasionally, however, evidence in the perimetric fields of pressure on the chiasm will be produced by tumors or inflammatory lesions at some distance from the optic chiasm. It is important to recognize this possibility, since an error in localizing the tumor may logically occur, particularly i the absence of other symptoms or neurologic findings which definitely indicate the location of the lesion.

In 1931, in a paper published in collaboration with Learmonth, Lillie and Kernohan [1] reported the finding of defects in the perimetric fields typical of prechiasmal and chiasmal lesions in a case of tumor of the vermis of the cerebellum and in a case of tumor of the acoustic nerve. In the first case, that of a boy aged 11 years, vision was reduced to 3/60 in each eye. Ophthalmoscopic examination revealed bilateral choked disk, the elevation measuring 2 D. in each eye. Studies of the

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1. Learmonth, J. R.; Lillie, W. I., and Kernohan, J. W.: Unusual Surgical Lesions Affecting the Optic Nerves and Chiasm, Am. J. Ophth. **14**:738-749 (Aug.) 1931.

visual fields on the perimeter showed large, bilateral cecocentral scotomas with bitemporal hemianopia for colors. At operation an astrocytic glioma of the vermis of the cerebellum was removed. The authors expressed the opinion that the ophthalmologic changes suggested a tumor of the third ventricle, a suprasellar tumor or a basofrontal tumor at the midline. The findings at operation and the postoperative course, however, showed definitely that the lesion was in the posterior fossa; the ventricular system was obstructed at that point. In the second case, that of a man aged 31 years, vision was 6/10 in the right eye and 6/30 in the left. Ophthalmoscopic examination revealed bilateral choked disk, the elevation measuring 5 D. in each eye. The fields of vision showed for the right eye inferior altitudinal anopsia for colors and for the left eye inferior altitudinal anopsia for both form and color. The clinical findings were those of tumor of the left acoustic nerve. At operation the tumor proved to be a perineural fibroblastoma of the eighth cranial nerve, but it was too extensive to be removed completely. At necropsy the tumor was seen to be confined to the posterior fossa. It had produced extreme compression and distortion of the cerebellum, pons and medulla oblongata, and moderate internal hydrocephalus without excessive pouching of the floor of the third ventricle.

Learmonth, Lillie and Kernohan gave no definite explanation of the mode of development of these changes in the fields of vision. They merely pointed out the possibility that an ophthalmologic syndrome suggesting direct pressure on the optic nerves or the chiasm may occur in cases of tumor in the posterior fossa.

In studying the fields of vision of patients with proved lesions in the posterior fossa, we have encountered a considerable number of cases of defects in the visual fields typical of a chiasmal or a prechiasmal lesion. We have found: (1) a central scotoma in each eye in one case; (2) a unilateral central scotoma with temporal hemianopia in the opposite eye in one case; (3) unilateral amaurosis with temporal hemianopia in the opposite eye in one case; (4) complete and congruous homonymous hemianopia in one case; (5) bilateral amaurosis without visible evidence of secondary atrophy of the optic nerve in one case, and (6) incomplete bitemporal hemianopia in one case. Ophthalmoscopic examination revealed chronic choked disks of varying degree in all the cases. Visible secondary atrophy of the optic nerve was present in each eye in two cases, and in one eye in another case. Ventriculography was performed in five of the six cases, and in all five marked dilatation of the third ventricle was noted. For this reason we began to feel that the defects in the fields might be attributed to pressure on the chiasm by the dilated third ventricle. This opinion was supported further by the findings in the following case.

REPORT OF A CASE

A man aged 38 years was admitted to the clinic on March 12, 1934. The history as given on admission was supplemented by additional data obtained subsequently. He was in good health until March 1930, at which time he began to have attacks of headache accompanied by gastric distress but not by vomiting. Several months later he was examined at a large diagnostic clinic, where a diagnosis of ulcer of the stomach was made. At that time bilateral, early choked disk was noted. Roentgenograms of the head showed no abnormality. A diet was prescribed, and for the succeeding ten months the patient was comfortable except for intermittent frontal headache.

In 1931 the patient again began to have gastric distress in spite of the use of alkalis. Headaches became more frequent and were accompanied at this time

Stretching of the optic chiasm and tracts by pressure exerted by the third ventricle.

by projectile vomiting. This continued intermittently for the next two years. In August 1933 he began to have attacks of unconsciousness; these lasted for about two minutes but were not accompanied by convulsions. In the latter part of 1933 the patient spent three months in a large general hospital. The diagnoses of peptic ulcer and inoperable tumor of the brain were made, and palliative treatment was advised. At this time bilateral, chronic choked disk, the elevation measuring 3 D. in each eye, was noted on ophthalmoscopic examination. The patient's vision was good. The headaches and projectile vomiting had continued. Six weeks before the patient came to the clinic his vision began to fail rapidly, and for three weeks he had been totally blind.

Examination at the clinic revealed marked weakness and cachexia, with moderate ataxia and a tendency to fall to the right. The patient appeared very ill. He was unable to distinguish light. Ophthalmoscopic examination revealed

bilateral, chronic choked disk, the elevation measuring 3 D. in each eye. No evidence of atrophy of the optic nerve was visible.

A diagnosis of unlocalized tumor of the brain was made, and the patient was advised to enter the hospital immediately. He returned to his hotel, however, and spent a restless night during which he vomited several times. Toward morning he became comatose and died before a physician could be summoned.

At necropsy the skull was observed to be very thin, and there was evidence of increased intracranial pressure. The third ventricle was markedly dilated and had a very thin wall which bulged about 2 cm. below its normal site. This downward pouching of the ventricle had markedly stretched and thinned the optic chiasm (as shown in the figure). The cerebellum was herniated into the foramen magnum. The internal hydrocephalus was due apparently to ependymitis, which had closed the foramen of Luschka and that of Magendie. The sella turcica was considerably eroded. No tumor was seen. Examination of the stomach showed no evidence of ulcer.

COMMENT

In this case choked disks were known to have been present for approximately four years. If the loss of vision had been gradual, it could have been explained readily by atrophy of the optic nerve fibers from long-continued edema. However, the loss of vision progressing to complete blindness in three weeks suggested the possibility of direct pressure on the optic nerves or chiasm, particularly in the absence of ophthalmoscopically visible evidences of atrophy of the optic nerve. That such pressure or its equivalent stretching was present was demonstrated definitely at necropsy. It was also demonstrated definitely that this pressure was exerted by the outpouching of the floor of the third ventricle. Anatomically, the anterior extremity of the third ventricle presents a diverticulum known as the recessus opticus, which extends forward above the optic chiasm. Dilatation of this recess can exert pressure on the chiasm similar to that of a suprasellar tumor or a tumor of the third ventricle. It seems logical to assume that dilatation of this recess may occur along with dilatation of the third ventricle proper as a relatively late effect of obstruction to the ventricular system in the posterior fossa.

It is important to recognize the fact that as a result of this mechanism changes in the visual fields typical of a prechiasmal or a chiasmal lesion may occur along with choked disks in the presence of tumors or inflammatory lesions in the posterior fossa. On the basis of the objective ophthalmologic examination alone it is often possible to differentiate this syndrome from that occurring in association with lesions in the anterior part of the brain. A carefully taken history of the onset of the loss of vision will often permit correct differential diagnosis. In the case of lesions located primarily in the basofrontal or the chiasmal region, loss of vision is always an early, and often the first, symptom noted by the patient. This loss of vision usually precedes by a definite interval the development of symptoms of general increased intracranial

pressure. In the case of lesions located primarily in the posterior fossa, on the other hand, subjective and objective changes in vision are late symptoms. Transient periods of blurring of vision may be associated with the development of choked disks, but permanent and progressive loss of vision associated with demonstrable changes in the fields of vision other than enlargement of the blindspots always follows by a definite interval the general symptoms of increased intracranial pressure, such as headache, vomiting and ataxia. In some of the cases of this type other clinical findings will point so definitely to the proper location of the lesion that the findings from examination of the perimetric fields may be disregarded. In other cases, however, localization of the lesion will seem to depend solely on the changes in the fields of vision. In such cases, even if the defects in the fields are typical of a chiasmal or a prechiasmal lesion, if there is a history definitely indicating an antecedent increase in the intracranial pressure it is wise for the ophthalmologist to advise ventriculographic studies rather than direct exploration of the basofrontal or the chiasmal region.

SUMMARY

A certain number of patients with a tumor or inflammatory lesion in the posterior fossa will show, in addition to choked disks, changes in vision and in the fields of vision which simulate those produced by tumors and inflammatory lesions in the basofrontal region or around the chiasm. These changes in vision and the fields of vision are late, not early, symptoms of lesions in the posterior fossa. They are due apparently to pressure on the optic nerves, the chiasm or the optic tracts near the chiasm by the dilated third ventricle. It is usually possible to distinguish these lesions from tumors in the anterior fossa by a careful taking of the history.

MUSCULAR ANOMALIES AND AMETROPIA

A PRACTICAL CONSIDERATION

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It may not be amiss to consider practically some of the questions that arise daily in the practice of the average ophthalmologist. For the younger ophthalmologists especially, who may not have met the unusual problems and who have been taught to follow the rules given in most works on refraction, the following remarks may be of some aid.

Rules are made to be broken, and the general rules are made more keenly interesting by a study of the exceptions to such rules. Not without a routine test of the muscle balance for distance and for near and with the eye in the six cardinal positions and a test of the powers of convergence and accommodation can unusual conditions be consistently found. Later, if and when glasses are prescribed, it is equally important to make tests to find if the glasses are doing what they are supposed to do or if by chance the condition has been made worse by following the prescribed rules. In any branch of medicine, if a certain course of treatment does not improve a condition the physician does not hesitate to change to other forms of treatment and to look for some complications which may alter or change a diagnosis. This is not done so frequently in cases of errors of refraction associated with muscular anomalies; rather, if the rules say to do so and so, this is done, and if improvement is not made it is the fault of the patient and not of the rules or of the physician who conforms to them. To emphasize these statements, a few conditions will be cited that have been seen either in patients dissatisfied with previous treatment or in patients seen in consultation.

When each patient examined for glasses is considered as presenting a potential problem of combined refraction, accommodation and muscle balance, the whole examination assumes a different aspect.

I wish to emphasize here the relief from the tedium of refraction that many ophthalmologists express and the satisfaction in finding the key to the problem, which has been overlooked by others, possibly not because of lack of knowledge but by failure to look in a routine manner for the less usual features.

Read before the Section of Ophthalmology of the New York Academy of Medicine, May 17, 1937.

Glasses may be given for one or more of three purposes: first, to improve vision; second, to relieve symptoms of asthenopia, headache and the allied symptoms, and third, to correct a muscle imbalance. If there is no muscle imbalance and the patient's only symptom is reduced vision, the question of prescribing glasses depends entirely on the patient's choice of glasses and good vision or of no glasses and reduced vision. The latter choice is made most commonly by women and girls who are myopic but who, having no other symptoms, choose, for cosmetic purposes chiefly, to allow the myopia to go uncorrected. A common theory is that myopia will increase more rapidly if uncorrected and that the accommodation will become progressively worse. With myopia up to 4 D, these results are rarely seen, and many myopes who frankly say that the glasses are worn only at the theater or occasionally for particular distant vision do not show more rapid progress of myopia, do not have the supposed loss of accommodation and do not acquire a muscle imbalance because of nonuse of glasses. A myope with vision of 20/200, which was corrected to 20/20, frankly said that he would use glasses only when clear distant vision was necessary. The correction caused no symptoms, but he did not like the sharply defined vision that the correction gave him. Being an artist, he pictured to me, when questioned, the haze of a late October day. If that is his pleasure and such vision is doing him no harm, why should correction of vision to 20/20 be arbitrary because of a theory? If, however, astigmatism is present, producing symptoms of asthenopia, if the accommodation is subnormal or if exophoria is present and is reduced or more easily controlled when the correction is worn, there is a reason for advising a more constant use of the correction, and full correction or even overcorrection may be beneficial. However, if a myope has esophoria which increases on the addition of minus lenses, it is often necessary to give an undercorrecting lens. Or the myopic correction may be used for distance and no glasses or an undercorrection of 1 or 2 D. used for near work. This may be best accomplished in some cases by the use of bifocal glasses, especially for children who are alternately changing vision, as from blackboard work to desk work.

Some form of exercises in divergence is beneficial in many cases of this condition, and such exercises definitely relieve the asthenopia, although the esophoria may remain the same.

Esophoria and esotropia in hyperopic patients constitute the muscle imbalance most frequently recognized. The patient is generally given a cycloplegic before a test has been made of the muscle balance or of the accommodation. These tests should be made before using a cycloplegic. Then a test of the muscle balance should be made with the eyes under the influence of a cycloplegic to determine the effect of the cycloplegic on the squint. If the cycloplegic has little or no effect on

the squint, little benefit may be expected from glasses for the correction of the hyperopia. If, however, the squint is definitely reduced by the cycloplegic, glasses will likewise reduce the deviation. After the correction has been worn and the accommodation has returned to normal, the muscle balance should be reexamined to determine the exact result produced by the glasses. If little or no improvement is made in two or three months by the use of a full correction, little benefit can be expected from the glasses.

If no other cause of the squint than hypermetropia has been detected in the earlier tests, a careful test should be made of the condition of the external and internal rectus muscles. In many cases in which the hypermetropia is less than 3 D. and especially in which the glasses have little, if any, effect, the real cause is due to hypertense internal rectus muscles or to underacting external rectus muscles. I reported before this section earlier the case of a child who had convergence of from 80 to 90 degrees, which was corrected by the recession of one internal rectus muscle. I think that, without exception, the internal rectus muscle was the largest that I have ever seen. The degree of hypermetropia was 1.50 D., and lenses did not affect the deviation in the least. Had the hypermetropia been greater and the muscle not so strong, the two factors might have combined to produce the squint, which would have varied in proportion to the relation between the two. However, many cases of this condition are observed in which glasses have been given to children as young as 1½ to 2 years of age and have been worn constantly for some years, with no appreciable effect on the squint. A safe rule would be that when correction of the hypermetropia fails, other or additional causes should be looked for.

More and more frequently I find either esophoria or esotropia, or exophoria or exotropia, associated with hyperphoria or hypertropia. The ametropia may be slight or considerable, may be myopic, hyperopic or mixed and may have some or no effect on the amount of deviation. These are the cases in which the results from the use of glasses or, in fact, from any form of treatment are the most disappointing, and there is little benefit until the vertical deviation is controlled surgically or otherwise.

Many of these patients have third grade fusion but much prefer to have either convergence or divergence. A girl aged 7 in one year showed a change from esotropia of 15 centrads for near, with no lateral deviation for distance, to exotropia of 20 centrads for distance, with no lateral deviation for near. There was right hyperphoria of 4 centrads due to a condition of a left superior rectus muscle. She had third grade fusion but preferred not to use it. Resection of the left superior rectus muscle was advised.

I have seen patients with the retraction syndrome and strabismus fixus associated with convergent strabismus wearing glasses in a forlorn hope that these may correct the deviation, and this hope was shared by the ophthalmologist.

Patients with exophoria and exotropia associated with hypermetropia vary widely in their response to treatment. If the theory of convergent strabismus that by relaxation of the accommodation with plus glasses the convergence is relaxed and causes the eyes to diverge held true here, one would expect that plus glasses for a patient with exophoria or exotropia would cause the eyes to diverge still more. This seems not to have occurred in the experience of many ophthalmologists, and it is not unusual to see a patient with exophoria or exotropia without glasses or a patient with esophoria or esotropia without glasses wearing the full correction, which made each condition a definite divergent squint. Such results can be definitely determined in the office, and more or less plus correction can be prescribed as indicated.

With this in mind, what should be done in the case of a Negro boy aged 5½ with marked divergent strabismus the amount of which was estimated by Hirshberg as 80 degrees and with vision of 20/70 in each eye as determined by retinoscopic examination with the eyes under the influence of atropine, the correction for the right eye being a + 8.50 D. lens ⊃ + 0.50 D. cyl. and that for the left being a + 9.50 D. lens ⊃ + 0.50 D. cyl?

One has to consider the racial and personal characteristics. The hypermetropia was so great that the patient became discouraged and refused to make any accommodative effort. A reduction of 2.5 D. gave apparent, although not actual, correction of the squint. An intense fighting personality, as often seen in the Irish, would have produced as great a convergent squint with the same amount of hypermetropia. The personal equation should always be carefully considered.

The personal equation is most important in the correction of presbyopia. I believe it was von Graefe who said "The younger the ophthalmologist, the stronger the addition given the presbyopes." Here personal habits, desires and business necessities should be considered carefully before one judges the strength of the addition to be prescribed.

Before a postgraduate group I suggested that a prepresbyopic ophthalmologist should decide on the strength of the addition without being prejudiced by knowing the patient's age. A person who is not presbyopic cannot appreciate the inconvenience experienced by too strong an addition for near. This depends more on personal preference than on actual age or accommodative powers. A patient aged 75 has accepted for years nothing stronger than a + 1.50 D. lens added to his full correction for distance.

Hypoclosis is frequently overlooked unless a routine test of the accommodation is made. Such was the case in a man of 26 who had marked asthenopia in vision for all near work. Vision was normal with the correction of slight hyperopic astigmatism. The muscle balance was normal except for a slightly remote convergence near point. The accommodation with his correction was from 2.5 to 3 D., with a typical fatigue reaction. Addition of a + 1.50 D. lens brought the accommodation for the near point to 4 + D. This was prescribed in temporary grab fronts to be worn for one week. The symptoms were entirely relieved, and bifocal glasses of the same strength were prescribed. This correction has been worn for two years, with no return of symptoms. The hypoclosis was probably due to a streptococcic infection two years before the patient's first visit, as the asthenopia dated from this illness.

SUMMARY

The kind and amount of ametropia should be determined approximately before the giving of a cycloplegic.

Tests of the accommodation and the muscle balance should likewise be made before a cycloplegic is given.

Tests of the muscle balance and frequently the test of accommodation should be made while the eyes are under the influence of a cycloplegic.

Then, with the eyes under the influence of a cycloplegic, the exact amount of ametropia should be determined.

With these data before one, rules are not necessary, but the condition in each case can be determined on its own merits, depending on the results of the various tests.

Such a procedure sounds more time consuming than it really is, but the results of the time so spent are most satisfactory.

LEVER ACTION OPERATION FOR INTRACAPSULAR EXTRACTION OF CATARACT

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Lever action intracapsular extraction of cataract is a new method; it derives its name from the fact that the lens in its capsule is dislocated and delivered by lever action. The scientific principle of lever action has been employed ever since the time of Archimedes, in 300 B. C., to perform work in which great resistance is offered or to raise a heavy weight with the expenditure of the minimum of power or effort.

In this procedure the lens is anchored by the attachment of its zonule. Some sort of power or effort must be applied so that the lens in its capsule may be dislocated by overcoming the resistance of the zonule. The nature of the power or effort applied in the different methods has been different. The power or force has to be applied directly to the lens or its capsule in some methods and from the outside only in others.

Thus, in Smith's method the power is applied as pressure with the lens hook from the outside on the sclera. This external pressure raises the tension of the vitreous, which breaks the resistance of the zonule. Traction with a spoon, lens hook or loop directly on the lens in its capsule is used in Pagenstecher's method. Traction on the coagulated lens by means of the diathermy needle is employed in Lacarrère's method.

Vacuum suction on the lens in its capsule is used in Barraquer's, Hulen's and Moore's method. Vacuum suction combined with pressure with the lens hook is employed in Green's method. Traction with the capsule forceps combined with pressure from the outside is employed in the Knapp-Török-Elschnig, Verhoeff, Stanculeanu and Sinclair methods.

In all these pressure, traction or suction methods and their combinations the force available for overcoming the resistance of the zonule is directly proportionate to the amount of effort or power employed. But at times the zonule is so firmly attached that the power required to dislocate the lens in its capsule is too much for the eye to bear.

On the other hand, with the lever action method the force available for overcoming the resistance of the zonule is greater in proportion to the amount of effort or power employed. Hence lever action intra-

capsular extraction is the safest of all the existing methods for removal of cataract.

Two silver instruments have been devised for the purpose of lever action: (1) the Hata-Donga (hyalonavicular) bisocketed fulcrum and (2) the mango leaf dislocator.

A lever has been described, in statics, as a rigid bar, straight or curved, which is capable of turning freely about a fixed point of support. This point of support is called the fulcrum. The effort or power is a force applied at any point of the lever so as to turn it about the fulcrum and thus to overcome a resistance or to raise a weight attached at any other point of the lever.

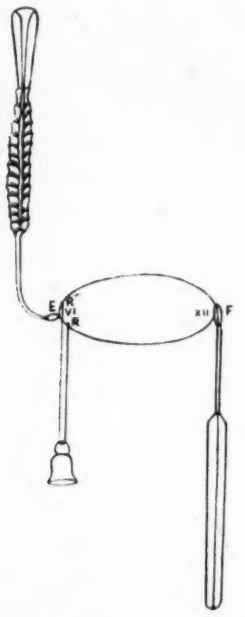

Fig. 1.—Diagram illustrating a lever of class II in the lever action operation for extraction of cataract. *F* indicates the fulcrum; *E*, the power or effort, and *R* and *R'*, the points of resistance or weight.

In the mechanism of the lever action operation for intracapsular extraction of cataract the lens, which has become more or less rigid by the formation of cataract, forms the lever. The resistance of the zonule, attached to the cataractous lens, must be overcome. This is done by fixing the edge of the equator of the lens at the uppermost point, viz., 12 o' clock, by giving the support of the hyalonavicular fulcrum. The effort or power is applied at the diametrically opposite, lowermost point of the equator of the lens, at 6 o'clock, by the mango leaf dislocator so that the cataractous lens in its capsule can turn about the fixed point of support in the hyalonavicular fulcrum.

In a lever of class II, the resistance or weight is placed between the fulcrum and the power or effort.

A lever of class II always acts at a mechanical advantage which is greater than unity.

The hyalonavicular bisocketed fulcrum is a silver instrument which consists essentially of two socket fulcrums: the hyaloid socket fulcrum and the navicular socket fulcrum, carved into the two surfaces of a silver plate. The hyaloid socket is of the shape and size of the hyaloid fossa of the vitreous, measuring about 5 mm. The navicular socket is a boat-shaped socket about 5 mm. long and is made in imitation of a 5 mm. section of the curve of the socket within the ciliary body, into which the equator of the lens in its capsule is socketed by nature. From this socket plate extends a rounded silver rod about 40 mm., long which ends in the handle of the instrument. The handle is a quadrangular silver bar 4 by 2 mm. in cross-section and 80 mm. long.

Nature has been imitated in the design of the instrument in order that lever action can be well enacted. The shape and size of the navicular socket being like the shape and size (5 mm. long) of the socket within the ciliary body, the uppermost margin of the lens in its capsule just fits into the navicular socket fulcrum, and cannot slip when in action, during the first and second stages of the operation. Similarly, the hyaloid socket fulcrum, being like the hyaloid fossa, receives the anterior surface of the lens in its capsule during the third stage of the operation and does not slip.

The mango leaf dislocator is a silver instrument which presents the following parts: a handle, a rod, a bend and a leaf-shaped tip.

The handle is made of silver plate and is 80 mm. long, 10 mm. broad and 2 mm. thick. About three fourths of the two faces is corrugated. A thin straight silver rod 40 mm. long is attached. The rod bends for 10 mm. at a rounded angle of about 90 degrees. The tip of the instrument is leaf-shaped, from which fact it derives its name. The leaf-shaped span is about 4 mm. long and is 3 mm. wide at its widest part. The tip is blunt and about 1 mm. thick. The blunt point cannot penetrate into the bulb of the eye when used on the surface of the eye from the outside.

TECHNIC OF THE OPERATION

After the usual procedures to insure antisepsis and induce anesthesia, a limbal incision into the upper half of the sclerocornea is made. Then the hyalonavicular fulcrum is placed at 12 o'clock on the outer surface of the sclera, and, the lens in its capsule is dislocated by fish-angling jerks applied at 6 o'clock with the mango leaf dislocator from the outside of the sclera. The lens in its capsule is delivered in three stages. The eye is kept bandaged for seven days.

Stage 1 (Dislocation of the Lens in Its Capsule).—The navicular socket is applied at 12 o'clock on the surface of the sclera, outside the insertion of the equator of the lens, and is kept fixed there. The portion of the sclera corresponding to the insertion of the lens in its capsule in the ciliary body can be easily made out, because the position is just behind the periphery of the insertion of the iris, which is visible through the operative cut. The navicular socket should be applied horizontally and tangentially to the optic globe. Thus, the navicular

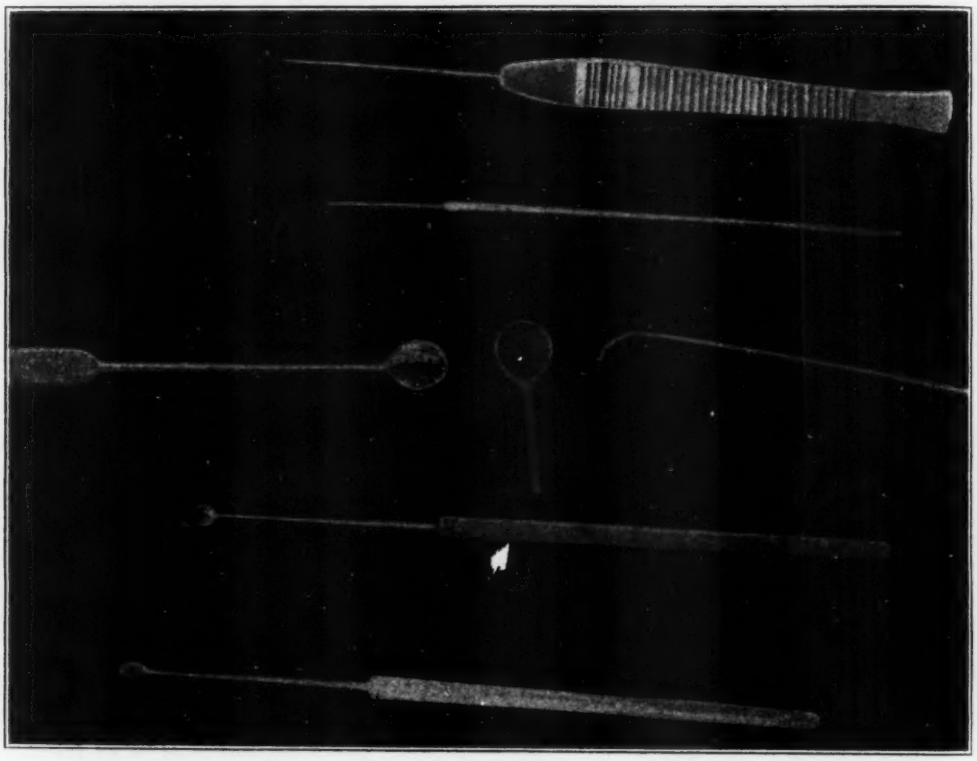

Fig. 2.—*1,* front view of the mango leaf dislocator, one-half the original size; *2,* side view of the mango leaf dislocator, one-half the original size; *3,* navicular face of the hyalonavicular fulcrum, twice the original size; *4,* hyaloid face of the hyalonavicular fulcrum, enlarged ×2; *5,* side view of the mango leaf dislocator, original size; *6,* hyaloid face of the hyalonavicular fulcrum, one-half the original size; *7,* navicular face of the hyalonavicular fulcrum, one-half the original size.

socket forms the fulcrum, into which is socketed the uppermost margin of the equator of the lens in its capsule, padded by the sclera. The lens in its capsule is thus capable of turning in the navicular fulcrum. The navicular socket fulcrum acts automatically, being of the shape and size of nature's own socket.

Now the effort or power is applied as fish-angling jerks by the tip of the mango leaf dislocator at the point diametrically opposite to the point of fixation of the fulcrum. That is to say, the jerky efforts are given at 6 o'clock. These jerks are given from behind forward, on the surface of the sclera, so as to hitch the equator of the lens in its capsule at 6 o'clock. The direction of application of power is therefore away from the vitreous. The jerks are given from 3 mm. behind the limbus at 6 o'clock forward. The tip of the mango leaf dislocator automatically hitches the more or less rigid lower border of the lens in its capsule so as to turn it forward, the navicular socket fulcrum keeping the upper border of the lens in its capsule fixed. These fish-angling efforts act on the resistance of the attachment of the zonule at 6 o'clock by lever action till the resistance is overcome and the zonule is breached at 6 o'clock.

Stage II (Rotation of the Lens in Its Capsule).—After the zonule is breached at 6 o'clock, the lower border of the lens in its capsule is tilted

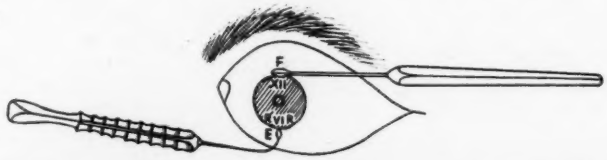

Fig. 3.—Diagram illustrating stage I of the operation, or dislocation of the lens in its capsule. *F* shows the navicular fulcrum, applied at 12 o'clock outside the sclera; *E,* the effort or power applied by the tip of the mango leaf dislocator at 6 o'clock outside the sclera, and *R* and *R'*, the points of resistance of the zonule to be overcome inside on each side of 6 o'clock. The more or less rigid cataractous lens is hereby converted into a lever. In this lever the resistance of the zonule is placed between the fulcrum and the effort; hence a lever of class II is formed (see figure 1).

forward and turns on the navicular fulcrum, which is kept fixed at the upper border of the equator of the lens in its capsule at 12 o'clock, and the second stage of the operation, or rotation of the lens in its capsule, begins. The navicular socket fulcrum is kept fixed on the outer surface of the sclera in the horizontal and tangential position, as before. The tip of the mango leaf dislocator is hitched below and behind the loosened lower margin of the lens in its capsule, the cornea intervening as a pad. By drawing the lower border of the lens in its capsule forward, away from the hyaloid fossa of the vitreous, and tilting it upward with the tip of the mango leaf dislocator, while the upper margin of the equator of the lens in its capsule, padded by the sclera, is fixed within the navicular socket fulcrum, the resistance of the zonule is easily breached by lever action.

The effort required to overcome the resistance of the zonule is less than the strength of the attachment of the zonule. This is because a lever of class II is formed in the mechanism, in which the resistance of the zonule is placed between the fulcrum, at 12 o'clock, and the effort, at 6 o'clock.

In this lever of class II the power or effort, EE', and the weight or resistance at the points R and R' or R'' and R''' act on the same side of the fulcrum, F, but the effort acts at a greater distance than the resistance from the fulcrum.

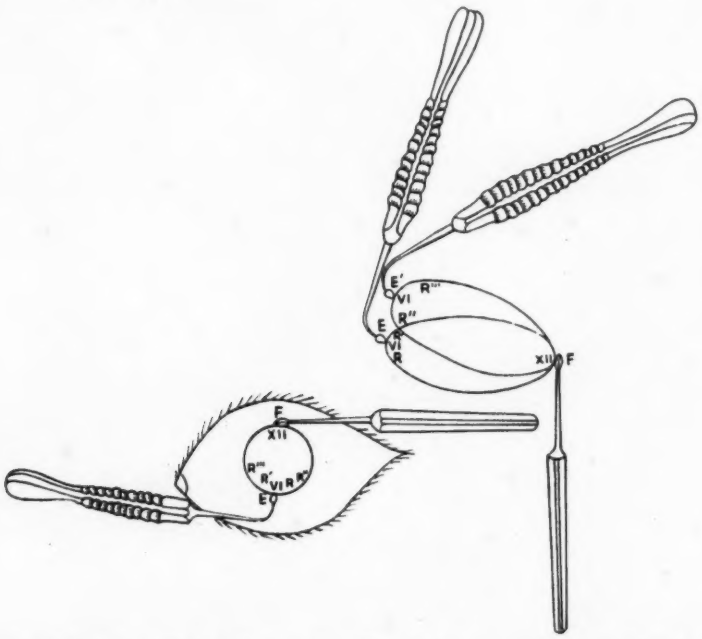

Fig. 4.—Diagrams illustrating stage II of the operation, or rotation of the lens in its capsule. *F* indicates the navicular fulcrum applied at 12 o'clock, socketing the upper border of the lens in its capsule; *E*, the effort applied steadily forward by insinuating the tip of the mango leaf dislocator at 6 o'clock on breach of the zonule from the outside to hitch the lower posterior border of the equator of the lens in its capsule so as to tilt it forward, and *R* and *R'*, the points of resistance in the attachment of the zonule. When *R* and *R'* give way, the resistance recedes to the points *R''* and *R'''*. The lower border of the lens in its capsule is rotated gradually forward and upward as more and more of the zonule is peeled off.

$$\text{Mechanical advantage} = \frac{FE}{FR \text{ or } FR'} \text{ or } \frac{FE'}{FR'' \text{ or } FR'''} = \frac{\text{Arm of effort}}{\text{Arm of resistance}}.$$

The arm of effort is the perpendicular distance between the fulcrum and the line of action of the effort or power = FE FE'.

The arm of resistance is the perpendicular distance between the fulcrum and the line of action of weight or resistance = FR, FR', or FR'', FR'''.

In a lever of class II, FE is greater than FR and FR′, and FE′ is greater than FR″ and FR‴.

Hence $\frac{FE}{FR}$ or $\frac{FE}{FR'}$ is greater than unity.

Therefore, the mechanical advantage in the lever action operation for intracapsular extraction of cataract is always greater than unity. Again, as more and more of the zonule is breached, the points of resistance at R and R′, which at first are at, or close to, 6 o'clock, just beyond which is the point of application of the effort, E, approach nearer the fulcrum, F, at 12 o'clock and move to the point R″ and R‴. Thus the arm of resistance FR″ and FR‴ becomes lesser and lesser than FR and FR′.

Therefore, $\frac{FE'}{FR''}$ or $\frac{FE'}{FR'''}$ is greater than $\frac{FE}{FR}$ or $\frac{FE}{FR'}$.

So the mechanical advantage becomes greater and greater as more and more of the resistance of the zonule is broken. Thus the effort by the mango leaf dislocator required to break the resistance of the zonular attachment is from the first stage of the operation less than, and afterward even still less than, the strength of the attachment of the zonule.

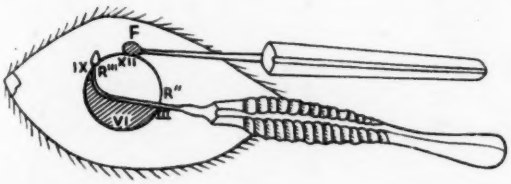

Fig. 5.—Diagram illustrating stage III of the operation. The hyaloid socket fulcrum is applied at 12 o'clock, and the bend of the mango leaf dislocator is applied behind and below the lower border of the lens in its capsule. The remaining portion of the zonule from 6 to 12 o'clock from the points of resistance *R″* and *R‴* up to 12 o'clock is detached gradually as the cataractous lens in its capsule turns on the hyaloid socket fulcrum.

A firmly attached zonule gives way safely and surely on the application of less effort in lever action intracapsular extraction.

As the lower border of the lens in its capsule rotates forward and up to an angle of 45 degrees, i. e., half a right angle on the fixed point of the cataractous lens at 12 o'clock within the navicular socket fulcrum as the result of effort applied at or about 6 o'clock with the mango leaf dislocator, the lower border of the lens in its capsule clears itself of the attached lower half of the cornea, and the third stage, or detachment of the zonule, begins.

Stage III (Detachment of the Zonule).—The lower border of the lens in its capsule has cleared itself of the attached portion of the cornea and has reached the operative cut. The hyaloid socket fulcrum is now placed on the anterior surface of the lens in its capsule. As the lens in its capsule has come out in the open operative wound, the anterior

surface of the lens in its capsule is in direct contact with the hyaloid socket fulcrum. The bend of the mango leaf dislocator is now applied below and beneath the lower border of the lens in its capsule to its posterior side. As the lower border of the lens in its capsule has cleared itself of the attached portion of the cornea, there is no longer an intervening pad of cornea between the lens in its capsule and the mango leaf dislocator. The bend of the mango leaf dislocator is now placed in direct contact with the posterior surface of the lower border of the lens in its capsule. A turning movement is given by the mango leaf dislocator forward and upward to the lower border of the lens in its capsule.

Now that the anterior surface of the lens in its capsule is securely fitted within the hyaloid socket fulcrum and cannot slip and at the same time turning effort is applied to the lower border of the lens in its capsule from the posterior side forward and upward by hitching the bend of the mango leaf dislocator, the lens in its capsule is easily turned forward and upward. Consequently the attached portion of the zonule is detached from below upward completely. The delivery of the lens in its capsule is completed by taking it out at the temporal side of the operative wound.

COMMENT

It may be asked: Why should a new method for the extraction of cataract be launched? What is the necessity of a new method in an era of capsulotomy and of numerous methods of intracapsular extraction? Are there not already so many methods of operation for cataract that the surgeon is sometimes much concerned about which method will be most appropriate in a particular case of cataract?

Those readers who have visited India are aware of the fact that at times the surgeon there has to operate on several hundred persons with cataract in a single day. It is well known that at least 100,000 persons with cataract are operated on in India every year. And still there remain some 500,000 reserves. It will thus be evident that the benefit of extraction of cataract has not yet been universally bestowed, at least in India, and that there are yet hundreds of thousands of human beings living in a state of partial blindness which is curable.

This state of things must be remedied. But how? By popularizing operation for cataract and by enabling the surgeon to operate successfully as soon as there is obstruction of sight.

In order to popularize operation for cataract it must be made safer and surer. Not only this, but it must be made easier, so that a surgeon of ordinary merit can operate well. In order to insure success of the operation at an early stage of formation of cataract, the cataract must be removed intracapsularly. In order to operate by the capsulotomy

method on a cataract, one has to wait till the cataract is mature. Thus the patient has to wait, living in partial blindness, for months or years. Some of the patients may not live long enough to receive the benefit of extraction of cataract. Living in partial blindness, unable to earn a living, bowed down with the weight of cares and anxieties, the patient with cataract goes to an early grave, cursing his ill luck.

This is one of the reasons why I set my heart on finding some solution of the unfortunate problem, a new method of operation by intracapsular extraction which might be universally applicable.

The next question is whether lever action intracapsular extraction answers the purpose for which it is ostentatiously launched. What is there in it to court and claim universality?

In the evolution of surgical intervention for cataract, ever since 1000 B. C., from which date up to the present time there are authenticated records, various methods have evolved. Of these, some have had their day and had to give place to newer and more satisfactory methods. Others are being practiced with more or less success at the present time.

Thus, the depression method of couching anterior route, inaugurated and practiced by Suśruta,[1] the father of surgical intervention for cataract, with his Yabamukhi Salaka (barley-tipped needle),[2] in which the lens was couched toward the lower inner region of the orbit, had no competitor for a thousand years. In 10 A. D. Celsus, of Rome, introduced his reclination method of couching by the posterior route.[3] But the vitreous was more damaged by the reclination method of couching than by the depression method. Therefore, in 300 A. D., Bagbhat, professor of surgery in Taxilla University, revived the depression method with certain modifications.[4] About 800 A. D. the califs of Bagdad had "Suśruta Sanhita" translated from Sanskrit into Arabic and named it "Ketaba Sushrud." The work both in the original Sanskrit and in its Arabic translation is read as a textbook. Couching was widely practiced till 1748, when Daviel, of France, originated the capsulotomy method.[5] A century later, Alexander Pagenstecher, of Wiesbaden, introduced extraction of the lens in its capsule with a spoon.[6] MacNamara practiced intracapsular operation by pressure of

1. Suśruta Sanhita (dated 1000 B. C.), vol. 7, chap. 17.

2. Mookerji, Girindranath: Surgical Instruments of the Hindus, Calcutta, Calcutta University, 1914, vol. 2.

3. Celsus, cited by Adams, W.: A Practical Inquiry into the Frequent Failure of the Operations of Depression and of the Extraction of Cataract, London, Baldwin, Cradock & Joy, 1817.

4. Bagbhat: Astanga Hriday, Calcutta, B. L. Sen, 300 A. D., vol. 8, chap. 14.

5. Daviel: Sur une méthode de guérir la cataracte par l'extraction du cristalin, Mém. Acad. roy. de chir., Paris **2**:337, 1753.

6. Pagenstecher, A.: Ueber Extraction cataractöser Linsen mit ihren Kapsel, Klin. Monatsbl. f. Augenh. **3**:316, 1865..

hand and published the pressure method in his book on ophthalmology in 1864. Smith popularized the pressure method which goes by his name and substituted the lens hook for the hand.[7] Barraquer, of Barcelona, introduced the vacuum suction method by his erisphake.[8] But Smith's pressure method and Barraquer's vacuum suction method, though practiced with success by the originator, were not so successful in other surgeons' hands. López Lacarrère has introduced the method of traction on the diathermically coagulated lens in its capsule.[9] Had the pressure method of Smith or the suction method of Barraquer been free from risks and complications, there would have been no need for further developments. But, as is well known, Smith's pressure method is attended with expression of the vitreous and Barraquer's suction method with suction of the vitreous and rupture of the capsule in a large percentage of cases.

Therefore, A. S. and L. D. Green combined Barraquer's suction method with Smith's pressure method in order to limit both suction and pressure. In the Knapp-Török-Elschnig, Verhoeff, Stanculeanu and Sinclair methods, traction with a capsule forceps is combined with Smith's pressure method.

With the induction of akinesia of the facial nerve by Van Lint's method and with the retrobulbar injection of procaine hydrochloride, many operators nowadays are able to operate by the combined methods. The operation is now more successful than before. But the prolonged intra-ocular instrumentation, though not immediately felt by the anesthetized eye, produces postoperative inflammation in many cases. So, although the operation is successful, the end-results are not quite satisfactory. And certainly it is the end-results that count. What is to be done?

All the possible combinations of pressure, suction and traction have been tried but have failed to attain the objective. What is left to do but to adopt some other form of application of force and to apply that force from the outside and in such a fashion that it does not press on or disturb the vitreous and the capsule in any way?

The lever action operation for intracapsular extraction answers the purpose. The effort is applied extra-ocularly and in a direction away from the vitreous. The vitreous is not pressed on or sucked. The capsule is not torn by traction or burst by suction. Hence lever action

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7. Smith, H.: Extraction of Cataract in Its Capsule, Arch. Ophth. **33**:64, 1904.

8. Barraquer, J. A.: Quelques indications de la phacoerisis, Clin. opht. **22**:387, 1917-1918.

9. López Lacarrère, J.: Die Electrodiafakie, Klin. Monatsbl. f. Augenh. **88**:778, 1932.

has a claim to universality. And, what is more, by the principle of lever action an extra amount of force is gained to the surgeon's advantage.

Now that I have operated on 300 patients with cataract without rupture of the capsule in a single case and with loss of vitreous in but 1 case, the lever action operation for intracapsular extraction is ready for respectful presentation to ophthalmologists and especially to my young friends. As regards the veterans, no doubt they can operate by any and every method. I told a veteran friend of mine who can boast of having operated on more than 100,000 patients with cataract and has a record of 700 operations in a busy day, "You have magic fingers."

For further particulars about the fish-angling lever action operation for intra-capsular extraction of cataract the reader is referred to the proceedings of the Fifth Annual Conference of the All-India Ophthalmological Society, to be published in September.

EFFECTS OF DINITROPHENOL ON THE PERMEABILITY OF THE CAPSULE OF THE LENS

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AND
M. L. TAINTER, M.D.
SAN FRANCISCO

In connection with clinical reports that cataracts occurred in patients who had previously taken dinitrophenol for reducing weight it became of interest to determine whether this drug could alter the permeability of the capsule of the lens in vitro and in vivo. If it were to be assumed that there is some connection between the use of dinitrophenol and the production of cataract it would be necessary to establish some rational mechanism to account for the phenomenon. One possibility, previously suggested by Krause [1] for other types of cataracts, is that dinitrophenol might decrease the permeability of the capsule of the lens and prevent the normal interchange of metabolites. On the other hand, it might increase the permeability so as to allow foreign materials to penetrate into the lens. Such hypotheses have led to fruitful investigations of other types of cataracts, as in the recent study of Gifford and his collaborators,[2] and therefore were of interest in this study. In an attempt to test these possibilities experimentally, the permeability of the capsule of the lens under the influence of dinitrophenol was measured in two ways: (1) in vitro by the utrafiltration method described by Friedenwald [3] and (2) in vivo, with a variety of dyes.

PERMEABILITY OF CAPSULE IN VITRO

Method.—Beef eyes were dissected out at the time of killing of the animals and brought directly to the laboratory. The lens was carefully removed and the capsule separated by making a cruciate incision in the posterior part of the capsule and removing it in a single piece. The capsules were gently shaken with physiologic solution of sodium chloride (0.9 per cent) with repeated changes until all the adherent lens material had been removed. Each capsule was then arranged

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1. Krause, A. C.: The Biochemistry of the Eye, Baltimore, Johns Hopkins Press, 1934.

2. Gifford, S. R.; Lebensohn, J. E., and Puntenny, I. S.: The Biochemistry of the Lens: I. The Permeability of the Capsule of the Lens, Arch. Ophth. **8**:414 (Sept.) 1932.

3. Friedenwald, J. S.: Permeability of Lens Capsule to Water, Dextrose and Other Sugars, Tr. Am. Ophth. Soc. **28**:195, 1930.

to function as an ultrafiltration membrane by being tied over the flared-out end of a piece of 7 mm. glass tubing. Care was used in handling and tying, and a smoothly fitting membrane which did not leak was obtained. To this was attached, by means of a short piece of rubber tubing, a graduated 1 cc. pipet, the whole being held upright by a clamp attached to a stand.

The tube and pipet were filled with a 1:2,500 solution of vital red H. R. in a 0.9 per cent solution of sodium chloride. The membrane on the lower end dipped into a vial containing 20 cc. of the saline solution without the dye. The heights were so adjusted that exactly 10 inches (25.4 cm.) head of pressure was exerted on the membrane. Under these conditions small amounts of fluid and dye were ultrafiltered through the membrane, the rate of filtration varying with the permeability. The volume of fluid passing through could be measured from the fall of the level in the pipet, and the amount of the dye by reading the concentration in the vial colorimetrically against suitable standards. The solution of vital red in some of the tubes contained dinitrophenol in a range of concentrations, the osmotic effect being balanced by adding dinitrophenol in exactly the same concentration to the saline solution in the vial. In this way the only unbalanced forces on each capsule were the osmotic pressure of the vital red and the 10 inch head of pressure of the column of fluid.

In any given experiment eight such ultrafiltration tubes were set up together as a series. These were allowed to stand for twenty-four hours filled with the solution of vital red, to permit the membranes to become saturated with dye and to test for leaks. Then they were carefully refilled with fresh solution of dye, and the amount passing through in the next twenty-four hours was estimated colorimetrically. This value for each lenticular capsule was taken as its control, since the magnitude varied slightly with the inherent permeability of the capsule, its tautness and the area of the membrane exposed for filtration. Then the control solution was carefully removed and replaced by solution of vital red in the same concentration to which dinitrophenol had been added. The first and last tubes were refilled with the solution of vital red alone to serve as controls on the spontaneous change in permeability with lapse of time. The other six tubes were filled with the solution of vital red containing dinitrophenol in concentrations of 1:5,000, 1:25,000, 1:125,000, 1:500,000, 1:2,500,000 and 1:12,500,000, respectively. At the end of twenty-four hours the amount of vital red which had passed through the membranes under the influence of dinitrophenol was read in the colorimeter as before. Interference from the yellow color of the dinitrophenol in the colorimetric readings of the vital red was prevented by adding a few drops of concentrated hydrochloric acid, which changes this indicator to the colorless form. The amount of vital red filtered by each capsule was calculated first as the percentage of the preliminary control value for that capsule, and this value was then calculated as the percentage of the average for the two control capsules which were not exposed to the dinitrophenol. In this way a value was obtained which expressed the change in permeability due to the drug and in which any spontaneous or progressive changes in the capsules had been canceled out. After the period with dinitrophenol, the experiment was run for two additional twenty-four hour periods, the tubes being filled with solution of vital red alone, in order to observe the time required for recovery after the dinitrophenol. As the periods of recovery in all the latter experiments did not differ significantly from the periods for the controls, they need not be considered further.

Results.—The experiments just described were carried through ten times and the average permeabilities in the entire ten series calculated;

the results are shown in the table. It can be seen that there was apparently a small increase in the permeability of the capsules, the largest change amounting to a 28.2 per cent increase with the 1 : 5,000 concentration of dinitrophenol. The variability of the results was such that the probable error of the means averaged ± 7.4 per cent, so that there was good probability that the larger changes were not due to chance.

There was, however, one uncontrolled factor in these observations: The solutions of dinitrophenol were made up by successive dilutions of a stock solution, which consisted of a 2 per cent solution of dinitrophenol in a 1 per cent solution of sodium bicarbonate. The sodium bicarbonate was used to dissolve the dye by forming its soluble sodium salt. Such a solution has an alkaline reaction and undoubtedly made the capsules in the tubes containing sodium dinitrophenol more alkaline

Amounts of Vital Red Passed by Ultrafiltration Through the Capsule of the Lens with Various Concentrations of Dinitrophenol, Expressed in Average Percentages of the Values for the Controls

Conditions	Period	Percentages Filtered with Various Concentrations of Dinitrophenol					
		1:5,000	1:25,000	1:125,000	1:500,000	1:2,500,000	1:12,500,000
Dinitrophenol in physiologic solution of sodium chloride (average of 10 series)	Dinitrophenol present	128.2	125.6	116.5	113.2	114.0	109.9
	First recovery.....	116.5	122.2	111.7	101.9	100.6	101.0
	Second recovery.....	106.8	107.0	102.7	97.5	94.5	97.1
Dinitrophenol in phosphate buffer of p_H 7.0 (average of 8 series)	Dinitrophenol present	101.9	99.0	93.7	107.4	93.3	106.2
	First recovery.....	98.1	98.4	103.4	100.0	102.8	101.4
	Second recovery.....	89.8	95.9	96.6	98.8	105.0	107.3

than those in the tubes containing the control solution. There was, therefore, the possibility that the increases in permeability were due to a difference in p_H rather than to any specific action of the dinitrophenol. This point was readily checked by running eight more series, in which, instead of the saline solution, an isotonic sodium phosphate buffer of p_H 7.0 was used, thus insuring that both the capsules in the tube containing the control solution and those in the tubes containing solution of dinitrophenol had the same reaction throughout.

In the lower half of the table, where the average results of this second group of experiments are presented, it may be seen that all the results cluster around the 100 per cent value. All the observed differences were less than the probable errors of the means, so that it is fairly certain there were no changes in permeability produced by the dinitrophenol. These experiments, therefore, show that if the hydrogen ion concentration is kept constant, dinitrophenol, even in concentrations as

high as 1 : 5,000, does not change the permeability of the capsule of the lens in vitro, which suggests that changes could hardly be expected in vivo.

PERMEABILITY OF CAPSULE IN VIVO

No very satisfactory means of measuring directly the permeability of the capsule of the lens in the living animal are available. Recourse was had to a technic used for demonstrating increased permeability of blood vessels, that is, the injection of dyes. If dinitrophenol increases the permeability of the capsule of the lens, it should facilitate the penetration of dyes into the lens.

Two or three rats at a time were given an injection of 50 mg. of a given dye per kilogram of body weight intraperitoneally. One rat was retained as a control, and the remaining one or two animals were given 20 mg. of dinitrophenol per kilogram subcutaneously. The dose of dinitrophenol was repeated about four hours later, if the rats appeared able to tolerate it. The survivors were killed at the end of six hours, and the lenses of all the rats were removed and carefully examined for the presence of color. A total of forty-nine different dyes, comprising crystalloidal and colloidal and acidic and basic compounds, were thus studied.

Dinitrophenol penetrated into the lens in about one half of the rats given the injections, as could be seen by the faint yellowish color when the lenses were compared with those of the rats not receiving dinitrophenol. In no instance, however, did the test dye penetrate into the lens to a visible degree. There was, therefore, no evidence that dinitrophenol could increase the permeability of the capsule of the lens in vivo sufficiently to let the molecules of dye through. When similar tests are carried out in vascular regions, such as the skin, the dyes pass through the capillary walls and stain the tissues readily in areas of increased permeability.

SUMMARY

Application of dinitrophenol to the capsules of beef lenses in vitro failed to change the permeability of these membranes, as indicated by negative results with the ultrafiltration of a colloidal dye.

Injection of dinitrophenol in rats also did not increase sufficiently the permeability of the lenticular capsules in vivo to allow penetration of the lenses by a wide variety of dyes injected intraperitoneally at the same time.

Accordingly no experimental basis was demonstrated, either in vitro or in vivo, for the assumption that dinitrophenol may cause a change in the permeability of the capsule of the lens of the eye.

SIGNIFICANCE OF THE FOVEAL DEPRESSION

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DETROIT

Among vertebrates which are not too strongly nocturnal a local modification of the retina for maximal visual acuity is of widespread occurrence. The modified area (which is single except in some birds) may be small or extensive, a broad horizontal band across the fundus or a suprapapillar crescent, round or in the shape of a variously oriented oval, located centrally or temporally even as far as the periphery and on or slightly above or below the horizontal meridian.

The generic term for such a region of high resolving power is area centralis—misnomer though this name often is. An area centralis may be distinguished from the surrounding retina only by the visual cells being more numerous in it per unit of retinal area. When this massing of visual cells is marked, however, it is usual to find several accompanying adaptations: The visual cells are slenderized, to facilitate their aggregation; they are elongated, to enable them to maintain their volumes and thus their individual limens; they are connected in smaller numbers to bipolar cells, and these in turn are connected in smaller numbers to ganglion cells, so that, the absolute numbers of neurons being increased, local thickening of the nuclear layers occurs; and the ratio of rods to cones is more or less reduced, the rods often being entirely eliminated.

The area centralis at its highest development is thus a complex of adaptations all of which make for increased visual acuity. The cones, which mediate sharper vision because they are less summated in opticus fibers, take precedence over the rods. Their diameters are reduced, and they are tightly packed together. This results in a piling up of the outer nuclear layer, and the other layers are correspondingly thickened and still further augmented by an actual increase in the ratio of ganglion cells to bipolar cells and that of bipolar cells to visual cells.

In a strongly diurnal animal the entire retina may, of course, be organized like an area centralis in some other animal, and one may in this case speak of "universal macularity." On the other hand, the need for a preponderance of rods and a maximal neurologic summation prohibits a strongly nocturnal animal from having an area centralis of any sort, unless the eye is very large.

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The area centralis, then, represents a compromise. Animals may produce one if they can afford to devote a portion of the retina to vision of high acuity, yet must retain most of the retina in condition to afford vision of high sensitivity. The adaptations which comprise an area centralis are local phenomena for the simple reason that the same patch of retina cannot be built for the highest visual acuity and for the highest visual sensitivity at the same time.

It might seem that if an animal had evolved a "perfect" area centralis by following the aforementioned rules, there would be nothing that it could do to increase still further, locally, the intrinsic resolving power of its retina. The area may become a pure cone one, and each cone may have a private opticus fiber; but the cones must have *some* diameter and would starve the usual avascular retina if they became too long.

One actually finds that in those animals with the greatest reputations for visual acuity the area centralis is often not a smooth, slightly elevated region but contains a central depression which may be round or oblong and shallow or deep. This depression is the fovea centralis (or fovea temporalis) and is present in some fishes, many reptiles, most birds, and some primates, including man.[1] Where a fovea occurs, another obvious factor increasing visual acuity is sometimes added—intra-ocular color filters of retinal types are especially well developed here,[2] and it has long been believed that the depression itself constitutes an improvement over the simpler, afoveate area centralis.

The foveal depression is created by the centrifugal displacement of the retinal elements in the two neuronic layers, so that the inner nuclear, inner plexiform, and ganglionic layers are virtually absent. The displaced material produces a circumfoveal annular (in the primates, macular) eminence, and the centrifugal course of the elongated foot pieces belonging to the cones of the foveal bouquet creates the "layer of Henle," the fibers of which slowly become shortened and erect to form the more ordinary outer plexiform layer of the extra-areal portion of the retina.

The fovea is thus a "thin spot"; and this common definition calls attention to the attenuation of the retinal layers. It is this attenuation which has been traditionally regarded as the raison d'être of the foveal depression. One commonly reads that removal of tissue from the visual path of the foveal cones permits light to reach them unimpeded. Indeed, the beginner in ophthalmology is likely to assume that the superiority of

1. See the summary of Kahmann, H.: Ueber das foveale Sehen der Wirbeltiere: I. Ueber die Fovea centralis und die Fovea lateralis bei einigen Wirbeltieren, Arch. f. Ophth. **135**:265, 1936.

2. Walls, G. L., and Judd, H. D.: The Intra-Ocular Colour-Filters of Vertebrates, Brit. J. Ophth. **17**:641 and 705, 1933.

foveal vision is largely due to the excavation of absorptive tissue and to minimize the importance of the other adaptations of the area centralis, or macula.

This accepted theory is predicated on the assumption that the transparency of the retina is appreciably less than that of the vitreous. The perfect clarity of living retinal tissue in situ renders this dubious; but in any case, and particularly in the region which is, to begin with, one of high threshold, little would be gained by taking advantage of the difference in extinction [3] occurring in the thickness of retinal tissue removed as compared with that taking place in the equal thickness of vitreous which naturally fills in the excavation. Again, an afoveate area centralis is always thicker than the unspecialized surrounding retina—in spite of which it is obviously much superior in resolving power, indicating that the production of a thin spot as such is not very important.

Still more serious objection arises from a consideration of the profiles of the highly developed foveae of diurnal sauropsidans. The fovea of a lizard (fig. 1) or that of a bird (fig. 2) is characteristically of what one may call the convexiclivate type, while that of man has an extreme concaviclivate form (fig. 3). The temporal fovea of birds, which is never so well developed as the nasal (central) one, may be continuously concave also, though not as broadly so as that of man. It is easy to see which of these two forms of fovea is the superior—though of course it has not yet been shown here what form has to do with such superiority. Surely a hawk with a million foveal cones per square millimeter would not be expected to have a fovea inferior in all-round efficiency to that of man, and performance indicates quite the opposite. Shallow foveae occur in poor-sighted birds such as the woodpecker, and it is well known that the semidomesticated pigeon has a shallow and variable fovea and the domestic fowl and the owls practically none. In birds, then, shallowness is a mark of foveal degeneracy brought about by the development of nocturnality or of domestication. Similarly, among the reptiles, in which the convexiclivate fovea is customary, there is one form, Sphenodon (fig. 4), in which the secondary adoption of nocturnality [4] has resulted in the foveal depression becoming broadened, shallow and tending toward the concaviclivate type as a step on the path to possible eventual elimination.

In the light of the situations in Sauropsida, the human fovea is thus seen to be actually crude. It either has never been developed to a high

3. The more detrimental phase of such extinction would of course be scattering, due to optical heterogeneity, rather than absorption; but retinal tissue appears to be quite homogeneous optically.

4. Walls, G. L.: The Reptilian Retina: I. A New Concept of Visual-Cell Evolution, Am. J. Ophth. **17**:892, 1934.

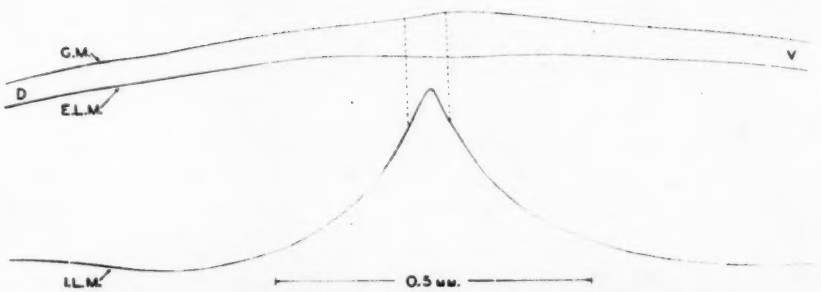

Fig. 1.—Microprojection tracing of the central section through the fovea of a diurnal lizard, Crotaphytus collaris. The portion of the retina between the dotted lines is thinner than the extrafoveal portion of the retina of the fundus generally. *G.M.* indicates the glassy membrane; *E.L.M.*, the external limiting membrane; *I.L.M.*, the internal limiting membrane; *D*, the dorsal side, and *V*, the ventral side.

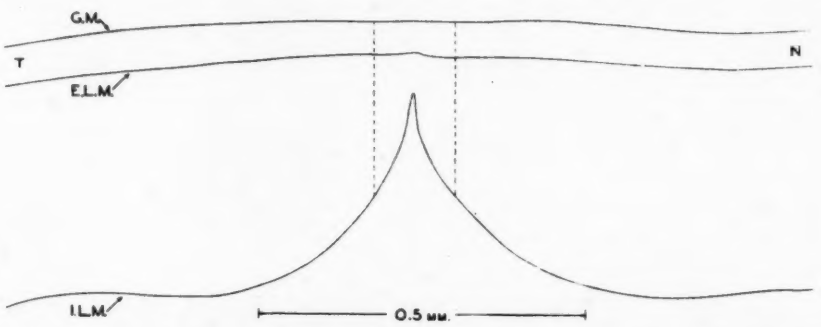

Fig. 2.—Microprojection tracing of the central section through the nasal (central) fovea of a hawk, Buteo b. borealis. The portion of the retina between the dotted lines is thinner than the extrafoveal portion of the retina of the fundus generally. *G.M.* indicates the glassy membrane; *E.L.M.*, the external limiting membrane; *I.L.M.*, the internal limiting membrane; *N*, the nasal side, and *T*, the temporal side.

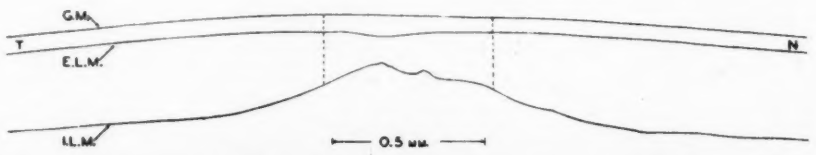

Fig. 3.—Microprojection tracing of the central section through a normal human fovea (from a slide loaned by Dr. Cecil S. O'Brien). The portion of the retina between the dotted lines is thinner than that in the extramacular portion of the fundus generally. *G.M.* indicates the glassy membrane; *E.L.M.*, the external limiting membrane; *I.L.M.*, the internal limiting membrane; *N*, the nasal side, and *T*, the temporal side.

degree or else it is degenerate.[5] Yet if attenuation were desirable per se, one would have to consider the convexiclivate form inferior and should be surprised not to find the foveal depression of the phenomenally sharp-sighted falcons and chameleons shaped somewhat as shown in figure 5. The portions of the foveae shown in figures 1 and 2 which are actually thinner than the respective retinas *outside* of the circumfoveal eminence[6] are proportionately far smaller in area than is the

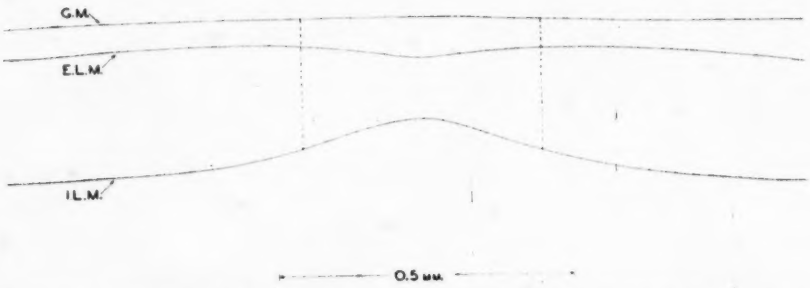

Fig. 4.—Microprojection tracing of the central section through the fovea of a nocturnal reptile, Sphenodon punctatus. The portion of the retina between the dotted lines is thinner than the extrafoveal portion of the retina of the fundus generally. *G.M.* indicates the glassy membrane; *E.L.M.*, the external limiting membrane, and *I.L.M.*, the internal limiting membrane.

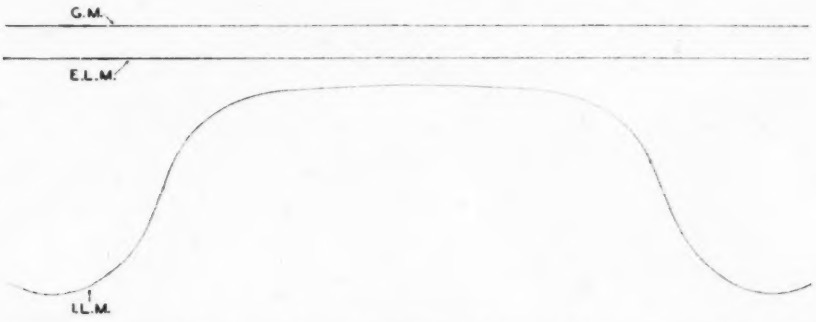

Fig. 5.—General form of foveal profile which would be expected in sharp-sighted birds, lizards and other species if the usual explanation of the foveal depression is assumed to be true.

5. It would be difficult to determine which, though a knowledge of the situation in the higher apes would help. The irregularities and variability of the human fovea are suspicious, however, for variability often accompanies degeneracy—as in the case of the fovea of the pigeon.

6. The term macular eminence is here avoided, since there is no macula in the strict sense. The much abused term macula lutea refers to coloration, not to conformation. Macular pigmentation is exclusive with the primates, though the avian foveal cones contain only yellow color filters and thus form a yellow spot with a different basis but with a similar significance for vision.

case with the fovea of man and that of Sphenodon (figs. 3 and 4). It is clear that the traditional theory of "removal of tissue" cannot explain the convexiclivate fovea. It is equally clear that one must temporarily dismiss the human fovea and fix attention on the superior sauropsidan type.

If the foveal clivus has some effect other than merely to attenuate the retina locally, that effect must be an optical one. The only optical performance which could affect vision and which can be assigned to the limiting membrane of the clivus is the constitution by that membrane of a refractive surface. The inclusion of the internal limiting membrane among the refractive surfaces of the eye has, naturally enough, never previously been entertained; but there is at least one phenomenon—the familiar foveal reflex of ophthalmoscopy—which might long since have aroused suspicion. The occurrence of this light reflex shows that either the internal limiting membrane or else the entire retina differs in optical density from the vitreous body. But the internal limiting membrane consists of Müller fiber substance, and if the Müller fibers were optically much differentiated from the rest of the retina there would be great detriment to vision. It thus seems logical that the general retinal tissue must differ from the vitreous body in refractive index.

If this difference were such that the retina had the lower index of refraction, total reflection in the deeper part of a convexiclivate fovea would render the central portion of such a fovea blind—which is unthinkable. There are, then, purely logical grounds for believing the retina to have a higher index of refraction than the vitreous body. For the retina generally, this will be of no consequence, because of the virtually perpendicular incidence of the perceived rays; hence there has never seemed to be need for experimental determinations of retinal refractive indexes, and such determinations have never been reported. In a well developed fovea, however, refraction at the clival surface takes on an importance which, in my opinion, is sufficient to account for the evolution of the foveal depression in the areae cantrales.

Figure 6 shows the optical effect of this refraction, a broadening of the retinal image between the two limiting membranes such that a much larger number of visual cells are brought into play than would otherwise be the case. Thus there occurs at the fovea a still further increase in resolving power beyond that produced by the organization of the best possible afoveate area centralis. The increased length of the foveal cones, which was interpreted previously [7] as a compensation for

7. Walls, G. L.: Human Rods and Cones: The State of Knowledge, Arch. Ophth. **12**:914 (Dec.) 1934. This increase occurs in fishes, reptiles and man but apparently not in birds.

reduced diameter in order to maintain the threshold, may also compensate for the lessened illumination of the expanded image.

If the refraction in the central portion of a fovea were sufficient to increase by 50, 30 or even 20 per cent the number of cones over which the image is spread, it could surely be argued that a very real optical importance had been found for the clival surface and its conformation. In the following analysis, for the sake of this argument an increase of 100 per cent is assumed. If the fovea is assumed to be circular, there need to be determined: (1) to what angle the rays from the lens must be deflected at the clivus in order to double the area of the image at the level of the external limiting membrane [8] and (2) the retinal refractive index demanded by this amount of deflection.

When two circles differ in area by a ratio of 2:1, their radii differ by the ratio of $\sqrt{2}:1$. In figure 6, which shows the same fovea as is

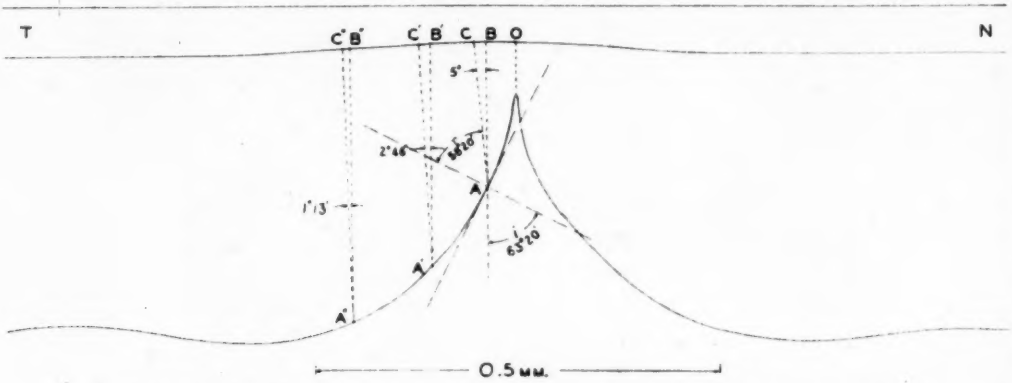

Fig. 6.—Semidiagram showing the enlargement of the retinal image by refraction at the clival surface of the fovea in accordance with the writer's theory. This is the same fovea as in figure 2, reconstructed on the basis of a plane glassy membrane. See the explanation in the text.

shown in figure 2, carefully reconstructed for convenience on the basis of a plane glass membrane, a point, A, has been chosen well down the clivus. A perpendicular from A to the external limiting membrane strikes the latter at B and represents the path that a perceived ray would take through the thickness of the retina if there were no refraction at the clival surface. When OC is laid off equal to $\sqrt{2} \cdot OB$ and AC is drawn, BAC is the angle of deflection required to double the area of an image the diameter of which is $2OB$ at the internal limiting membrane and the center of which is coaxial with O. This angle proves,

8. There are several reasons for believing that rays entering the bases of the visual cells are confined within the latter by total internal reflection. Hence for the present purpose the external limiting membrane may be taken as the level of perception.

on graphic solution in figure 6, to be 5 degrees. Tangent and normal lines have been drawn at *A*, and the angle of incidence, *i*, is found to measure 63 degrees and 20 minutes. The angle of refraction, *r*, is then 58 degrees and 20 minutes. With the use of 1.336 as the refractive index of the vitreous body in the equation $n_1 \sin i = n_2 \sin r$, the refractive index of the retina, n_2, is found to be 1.403.

Whatever the actual index of the retinal tissue, the increase in the size of the image naturally falls off as the image subtends more and more of the depression. A refractive index of 1.403 being assumed, the angles of deflection at *A'* and *A"* in figure 6 have been measured, the construction lines being omitted for the sake of clarity. *B'A'C'* is an angle of deviation of 2 degrees and 46 minutes, and the ratio *OB'*: *OC'* is such that there is a 29.80 per cent increase in the area of the image. The corresponding angle *B"A"C"* is 1 degree and 13 minutes, and the area of the image is increased 10.36 per cent. It is not proposed that any valuable deflection occurs at points between *A"* and the crest of the circumfoveal eminence.

It follows that an image confined to the fovea or the foveally perceived portion of any larger image is aberrated by the change in the angle of incidence along the clivus. More prominent aberrations than this are taken care of centrally and eliminated from subjective vision, however. The valuable obtuse angle in the deeper portion of the fovea cannot, of course, be formed abruptly by living tissue.

Though it is unlikely that the human (or any) retina has a refractive index as high as 1.4, and though the human fovea is concaviclivate, there should nevertheless be a valuable expansion of the image even in man. Even the shallow depressions ("incomplete" foveae—Kahmann) seen in some animals should at least have a dispersive effect sufficient to counteract the contractive action on the image of the otherwise convex surface of the area centralis. If one wonders that the majority of foveae, including human foveae, seem poorly formed for the best performance of the function here suggested, it should be borne in mind that most areae centrales are much inferior to those of average birds and lizards in all other respects as well.

It is to be hoped that investigators who have the proper equipment available will soon furnish data as to actual retinal refractive indexes. I am assured that there are applicable methods of determination and that there are no theoretical or practical difficulties which should be insuperable. If retinal tissue proves to have an index to any degree approaching the value of 1.4 stressed for the sake of argument in the foregoing reasoning, it may safely be concluded that the principal function of the foveal depression is the one proposed in this communication. Otherwise, a reasonable explanation of the depression is still to be sought, for the comparative situation shows the traditional theory of the "removal of tissue" to be untenable.

CHANCRE OF THE UPPER EYELID IN AN INFANT TWO MONTHS OF AGE

REPORT OF A CASE

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NEW YORK

Although the ocular region is not an uncommon site for extragenital chancres, chancre in an infant and involvement of the upper eyelid are both rare.

Marin [1] reported a case of chancres on the vulva in a girl of 11 months and another case of chancre of the upper lip in a child of 2 years. Foveau de Courmelles [2] reported a case of chancre of the gums in an infant. Bertin, Christin and Lesenne [3] reported a case of chancre of the conjunctival surface of the upper eyelid in a girl of 8 years.

Fox and Machlis [4] reported the case of a colored man who showed a chancre of the upper eyelid eight weeks after being bitten on that structure while fighting. T'Ang and Hu [5] reported a case of conjunctival chancre involving the middle portion of the upper fornix in a Chinese man of 43, who simultaneously presented a penile chancre. Gaté and Genet [6] reported a case of chancre at the internal angle of the eyelids in a man of 26.

Bulkley [7] classified 9,058 cases of chancre reported in the literature and found the eyelids and conjunctiva to be involved in 4 per cent.

Read before the Section of Ophthalmology of the New York Academy of Medicine, May 17, 1937.

1. Marin, A.: Deux cas de syphilis acquise chez le nourrisson, Union méd. du Canada **63**:374-377 (April) 1934.

2. Foveau de Courmelles: Infection syphilitique gingivale par une brosse à dents, J. d. mal. cutan. **17**:705, 1905.

3. Bertin, E.; Christin and Lesenne: Chancre syphilitique de la face conjonctivale de la paupière supérieure, Bull. Soc. franç. de dermat. et syph. **40**:213-215 (Feb.) 1933.

4. Fox, Noah, and Machlis, Samuel: Primary Syphilitic Lesion of the Upper Lid, Am. J. Ophth. **1**:701-702 (Sept.) 1924.

5. T'Ang, T. K., and Hu, C. K.: Chancre of Retrotarsal Fold, Nat. M. J. China **17**:106-109 (Feb.) 1931.

6. Gaté, J., and Genet, L.: Chancre syphilitique de l'angle interne des paupières, Bull. Soc. franç. de dermat. et syph. (Réunion dermat.) **41**:553-555 (April) 1934.

7. Bulkley, L. D.: Syphilis in the Innocent, New York, Bailey & Fairchild, 1894.

Subsequently Muncheimer [8] and Scheuer [9] classified 10,265 and 14,590 cases of chancre, respectively, and verified this percentage.

Maxey [10] studied the reports of a number of cases of primary syphilitic lesions of the eye and noted the frequency of occurrence shown in the tabulation. It appears that the conjunctiva, particularly the bulbar portion, offers Spirochaeta pallida biologic conditions especially conducive to its growth.

The case reported herein is of interest for several reasons: (1) the unusually early age at which infection occurred and the primary lesion appeared, (2) the maternal source of the infection and (3) the negative results of the initial dark field examination of serous material obtained from under the crust of the suspected, untreated lesion.

Site	Number of Cases
1. Conjunctiva	
Upper palpebral	5
Superior fornix	3
Lower palpebral	3
Inferior fornix	3
Location indefinite	13
Plica and tarsus	1
Bulbar	21
2. Lid	
Upper	1
Lower	4
Ciliary border	2
Location indefinite	27
3. Canthus	4
4. Location unknown	2

REPORT OF CASE

History.—W. C., a Puerto Rican boy born Jan. 6, 1937, was brought to the ophthalmic clinic of the Flower-Fifth Avenue Hospital on March 16, 1937. The mother stated that about March 6, two months after birth, she noted a swelling of his right upper lid. After the first few days the swelling began to discharge and then broke into an "open sore." As it increased in size, she became alarmed and sought medical attention. The infant's life was otherwise normal up to that time.

Examination (March 16, 1937).—The outer half of the upper lid of the right eye, the lower lid and the bulbar conjunctiva were perfectly normal. The inner half of the right upper lid, however, presented a sharply defined oval ulcer about 3 mm. wide with its long diameter on the horizontal level, mounted on an induration which appeared to be painless to manipulation. The surface of the ulcer was

8. Muncheimer, F.: Ueber extragenitale Syphilisinfektion, Arch. f. Dermat. u. Syph. **40**:191-235, 1897.

9. Scheuer, O.: Die Syphilis der Unschuldigen, Berlin, Urban & Schwarzenberg, 1910.

10. Maxey, E. E.: Primary Syphilis of Palpebral Conjunctiva, Am. J. Ophth. **1**:13-17 (Jan.) 1918.

uniformly level, dry, crustlike and about 0.5 mm. below the level of the overlying border of the slightly edematous encircling skin. At the free margin of the ulcerous lid was noted some dried-up secretion. The upper palpebral conjunctiva was somewhat injected. The preauricular glands on the right side were enlarged and palpable.

A, appearance of the chancre on the patient's admission to the hospital; *B*, appearance of the chancre one week after local and general treatment was instituted; *C*, appearance of the right lid after healing of the chancre, which left a notched defect in the margin of the lid.

A smear showed no organisms, but many pus cells were observed. Dark field examination of serous material removed from under the crust gave negative results. Specimens for both these tests were taken at the time of the first examination, when I strongly suspected a chancre.

Cleansing solutions were prescribed, and the mother was instructed to return with the infant on the next clinic day, March 19. On her return to the clinic the discharge at the margin of the lid was noted to be more moderate in quantity. Another dark field examination was ordered. This time spirochetes were observed. The child was isolated at the Metropolitan Hospital the next day.

Examination made at the hospital confirmed the diagnosis of chancre. Spirochetes were again demonstrated in the ulcer by dark field examination on March 20. The ulcer became moist and presented a brownish red crusty base. A serosanguineous discharge was present, and many spirochetes were noted in it. The injection of the upper palpebral conjunctiva persisted. General examination of the infant gave negative results excepting for the preauricular glands on the right side, which became markedly enlarged. The results of urinalysis and of a blood count were negative. The Wassermann test of the blood was strongly positive—four plus.

Treatment.—This was carried out in the isolation ward of the Metropolitan Hospital, New York, under the direction of Drs. Van Alstyne Cornell and Girsch Astrachan.

A 10 per cent emulsion of bismuth subsalicylate in oil was administered intramuscularly in three initial injections, in doses of 0.08, 0.1 and 0.13 cc., in the order stated at intervals of three or four days. This treatment was followed by the concurrent administration of mapharsen, injected intramuscularly, and bismuth subsalicylate in oil, injected intramuscularly, each given once a week for fifteen weeks.

The initial dose of mapharsen was 1 mg., and this was increased each week by 0.5 mg. until a dose of 5 mg. was reached; this dose was then maintained. The dose of the bismuth preparation was maintained uniformly at 0.17 cc.

The infant appeared to bear the treatment well and gained the usual weight. No untoward effects were noted at any time, and no complications resulted. While the chancre was completely healed at the close of this course of treatment, the Wassermann reaction of the blood remained unchanged.

The age of the patient, solely, made it unnecessary clinically to differentiate the lesion from a gumma. Yet it must be borne in mind that the latter may commence as an indolent tumor resembling a chalazion or a hordeolum but soon breaks down into an indurated, painless ulcer which tends to invade the deeper structures of the lid, sometimes going on to perforation of the tarsus.

The source of infection in this case is exceedingly interesting. With the cooperation of the Department of Health of the City of New York the following facts were disclosed:

The father of the baby had indulged in extramarital intercourse about a year and a half before, and a penile "sore" developed several weeks later. He received no treatment for five weeks and then placed himself under the care of a physician, on Dec. 10, 1935. The Wassermann reaction of his blood was found to be strongly positive—four plus. After being under treatment for one month, the father brought his wife to the same physician for examination. Her blood was found to show a three plus Wassermann reaction. During the period of Dec. 10, 1935, to Aug. 25, 1936 (eight and one-half months), the

father had received a total of fifteen injections of neoarsphenamine (0.6 Gm. each) and sixteen injections of bismuth subsalicylate in oil, at irregular intervals. The mother had received two injections weekly, bismarsen and bismuth subsalicylate in oil being given alternately, during the period of Jan. 10 to May 11, 1936 (until about the beginning of her pregnancy), and then she became irregular in her attendance. She had had only four injections of bismarsen and five of bismuth subsalicylate in oil during the entire prenatal period. The total amount of treatment received by her during the period of January 10 to August 25 included two injections of neoarsphenamine, fifteen of bismarsen and seventeen of bismuth subsalicylate in oil.

The records of the Flower-Fifth Avenue Hospital revealed the following points in connection with this case: The mother, aged 22, gave a history of one miscarriage of unknown date but no history of syphilis. The Wassermann and Kahn reactions of her blood on Aug. 12 and Sept. 30, 1936 (just before and after the midterm), were negative. On Jan. 6, 1937, she gave birth to a full term normal and healthy-looking child, 7 pounds and 5 ounces (3,317 Gm.) in weight. The placenta was intact, and the Wassermann reaction of blood from the cord was negative. No abnormalities were noted in the mother or the child on delivery or on discharge from the hospital. A follow-up record, dated three weeks after delivery, likewise showed no abnormalities in the baby, who at that time had gained 1 pound and 7 ounces (652 Gm.) in weight.

On February 2, about one month after delivery, a generalized cutaneous eruption developed in the mother. One month after this eruption she noticed what she described as the "sore" on the right upper eyelid of her baby. Examination on March 24 at the Meinhard Venereal Disease Clinic disclosed that she had a generalized papular cutaneous eruption, which was diagnosed as "late secondary."

COMMENT

Evidently this infant was infected by his mother, who, in turn, had probably contracted the syphilis from her husband. Furthermore, it appears that the infant profited from the limited antisyphilitic treatment received by his mother and was born free from manifestations of hereditary syphilis. Just when and how the infection occurred is a matter of conjecture.

The mother stated with certainty that neither she nor any one else ever kissed the baby; that the infant always enjoyed the use of a freshly laundered towel (not boiled) but she never set aside any towels exclusively for his own use, and that she had no lesions on her breasts and used a nipple shield when nursing the baby.

It is generally accepted that secondary lesions are infectious only when situated on surfaces of mucous membrane where biologic conditions are conducive to the growth of S. pallida, that drying almost immediately destroys the organism, that a break in the skin is not necessary for infection to occur and that the incubation period of a chancre may be as long as ninety days.

Taking into consideration all the facts and findings, I am led to the conclusion that the source of infection in this case was a secondary lesion present in the mother's birth canal at the time of delivery, a prolonged incubation period following the contact.

USE OF IODIZED POPPY-SEED OIL IN OPHTHALMOLOGY

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NEW ORLEANS

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Iodized poppy-seed oil, an opaque contrast medium, has been utilized for the roentgenologic exploration of various cavities and organs of the body, such as the bronchopulmonary apparatus, the spinal cord, the paranasal sinuses and the genito-urinary tract. The purpose of the present investigation was to determine the value of the iodized oil in the roentgenologic exploration of the tracts and cavities about the eye. At the same time an attempt was made to ascertain its usefulness as a therapeutic agent in various diseases of the eye.

Iodized poppy-seed oil is a definite chemical combination of iodine and poppy-seed oil. It is not a solution of iodine in poppy-seed oil but represents a stable chemical union between these two substances. The standard preparation contains 40 per cent pure iodine by weight yet does not give the ordinary tests for iodine. At room temperature the oil is viscid, but heating to 35 or 40 C. causes it to become much more fluid, a property which greatly facilitates injection.

During the past fifteen years a vast literature has accumulated dealing with various uses of iodized poppy-seed oil in diagnostic roentgenology. It is generally admitted that with very few exceptions the oil may be injected into practically every tissue or cavity of the body, causing little, if any, pain at the site of injection. In spite of this the instillation and injection of the iodized oil into the ocular tissues and spaces were done with the greatest of caution, because of the uncertainty of the outcome. At first the effects of instillation into the conjunctival cul-de-sac were observed and recorded. When it was established that this was a harmless procedure the oil was injected into the periocular tissues and finally into the vitreous.

INSTILLATION

At the beginning of the experiment the oil was dropped into the conjunctival cul-de-sac once daily for a period of several weeks. Sub-

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sequently this procedure was changed, and an instillation was made four or five times daily for many months, without producing any irritation or annoyance other than slightly blurred vision arising from the oily film on the cornea. The iodized oil did not accumulate in the folds, nor did it remain in the cul-de-sac in sufficient quantity to enable one to obtain a roentgenogram of the surface of the eye. The motion of the lids quickly removed any excess oil from the surface of the eye and thereby made it impossible to procure a roentgenogram. The oil did not irritate the conjunctiva or the cornea; on the contrary, it proved to be a pleasant demulcent.

INJECTION

After it had been determined that no unpleasant effects followed the instillation of iodized poppy-seed oil into the conjunctival cul-de-sac, injection was made into the stroma of the conjunctiva. Although the mesh of the tissue impeded the flow of the iodized oil and broke it into globules, no pain or untoward reaction ensued, in spite of the fact that the oil remained in the tissues for many months.

The next procedure was to inject the iodized oil into the spaces formed by the folds of orbital fascia about the eye. Here, also, the injection was practically painless, and no untoward reaction was produced. In addition, roentgenograms were obtained of the areas into which the injection was made.

Subsequently the oil was injected under the retina and also into the vitreous and anterior chamber. Wherever it was injected, whether into the conjunctiva, into the spaces formed by the orbital fascia, under the retina or into the vitreous, no discomfort followed. In certain tissues and cavities the iodized oil spread; in others it formed globules and gravitated to the lowest point. For instance, when injected under Tenon's capsule it spread out, and when injected into the vitreous it assumed a globular form. The preparation, being an oil, will not mix with the liquids in the vitreous or in the anterior chamber, while the space under the capsule represents a cavity with a pressure which is sometimes less and sometimes greater than atmospheric pressure.

The orbital tissues and spaces into which injection was made are shown in a schematic view (fig. 1). Other figures (figs. 2 and 3) are roentgenograms of the retrobulbar space and the cone of fat.

Into the Episcleral Space.—The injection of this space formed by Tenon's capsule reveals valuable information (figs. 2 and 3). The globe may be effectively visualized in this way, for both lateral and anteroposterior roentgenograms of the eye in its orbit may be obtained. The lateral view appears as if a horizontal section were taken through the eye and orbit. The anteroposterior view gives the appearance of arcs

of densities concentric with the axis of the eye. If an excess of the oil is put under Tenon's capsule, it may be seen coursing in the sheaths of the rectus muscles.

Into the Retro-Ocular Cone of Fat.—After injection of the oil the space of fat which is formed by the rectus muscles and the connective tissue between them resembles an opaque triangular plaque when viewed anteriorly, particularly when the eye is turned up or down, away from the plane. In the lateral view this is, of course, different in shape, conforming to the position of the eye.

Under the Retina.—When injected under the retina the oil does not spread out in a film but assumes a globular shape and travels to the lowest level of the eye. The intra-ocular pressure is not sufficient to

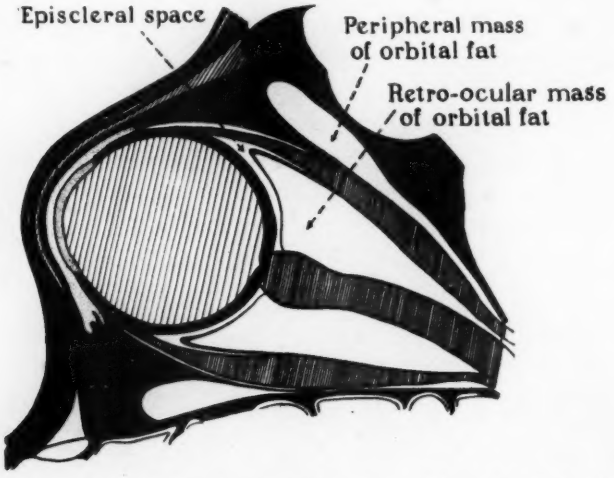

Fig. 1.—Schematic view of the tissues and spaces subjected to injection of the oil.

flatten this globule of oil, and furthermore the oil will not mix with the liquid under the detached retina.

In Cases of Detachment of the Retina.—Iodized poppy-seed oil was injected under the retina in two cases of retinal detachment. In the first case the subretinal fluid was not removed before injection. In the second case the fluid was removed with the hope that the oil would spread out instead of assuming a globular form at the lowest point. In each case, however, it remained in globular form in the eye for many months. Subsequently, multiple punctures were made through the sclera, and the retina became reattached.

Into the Vitreous.—Injections of iodized poppy-seed oil into the vitreous were first performed on rabbits. Only after it was demonstrated that this was innocuous were injections of the oil made into the human vitreous. The oil in the vitreous of the rabbit's eye is shown in figure 5.

After extraction of a cataract large quantities of vitreous were lost. The suture which had been inserted before the extraction was then tied, and the iodized oil was injected to replace the lost vitreous. However, the oil was retained in the eye with great difficulty, and that which remained did not seem to serve any useful purpose.

Fig. 2.—*A*, lateral roentgenogram of the cranium, showing to what extent the eye can be outlined in the orbit after the opaque oil has been injected into the episcleral space (Tenon's space). *B*, anteroposterior roentgenogram of the skull, showing arcs of densities concentric with the axis of the eye after the opaque oil was injected into the episcleral space (Tenon's space).

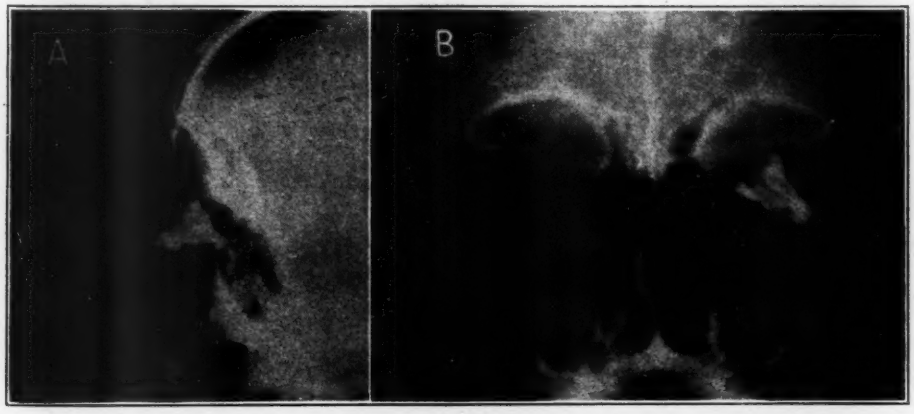

Fig. 3.—*A*, lateral roentgencgram of the skull, showing the retro-ocular space of fat after injection of the opaque oil. The apex is toward the bony optic foramen. *B*, anteroposterior roentgenogram of the retro-ocular space of fat after injection of the opaque oil.

IODIZED POPPY-SEED OIL AS A THERAPEUTIC AGENT

The most obvious benefit from the use of the iodized oil occurred in cases of blepharitis marginalis and in some cases of conjunctivitis.

Fig. 4.—Roentgenogram showing the oil under a detached retina.

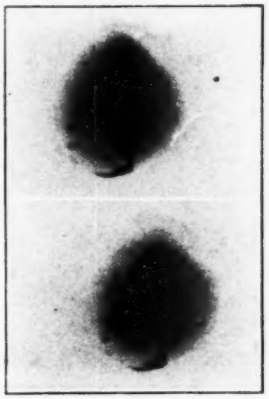

Fig. 5.—Roentgenograms taken after the iodized oil was injected into the vitreous.

In the cases of inflammation of the lid the scabs and crusts readily disappeared. In cases of pemphigus of the conjunctiva the oil proved most valuable, and recovery was expedited. Small corneal ulcers responded favorably, whereas larger ulcers showed no improvement.

Because of the large iodine content of iodized poppy-seed oil, patients with incipient cataract were treated with instillations of the oil into the conjunctival culdesac. The treatment was continued for many months

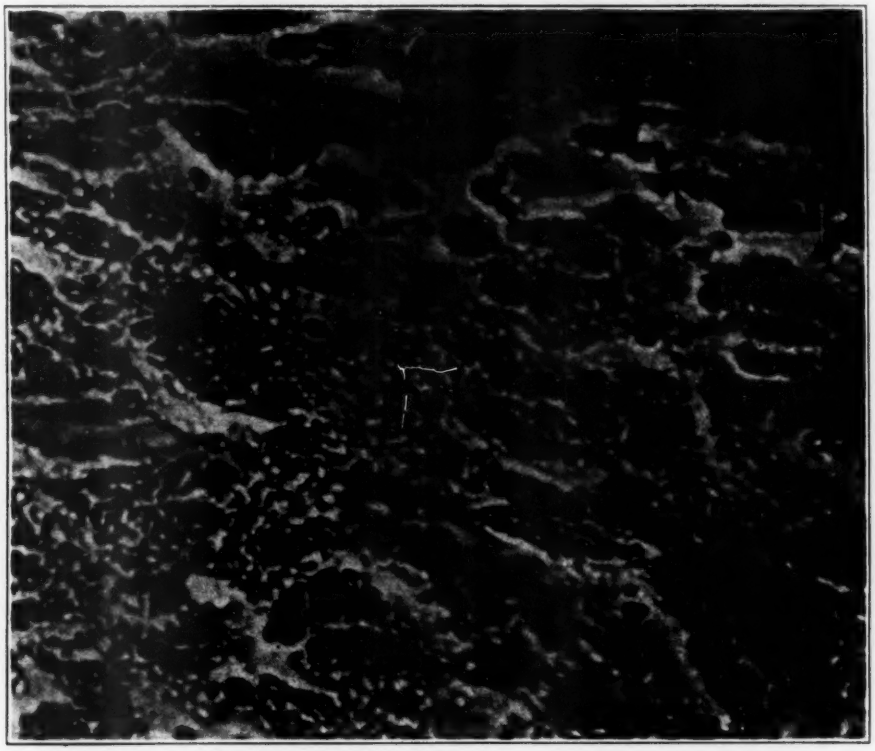

Fig. 6.—Histologic picture of the tissue reaction following the injection of the oil into the pterygium.

with the hope that the iodine would eventually be liberated. It is not quite possible to say that this occurred. However, in one case of detached retina, after a period of several weeks the injected oil changed in color. In another case the oil hastened the ripening of the lens. In a previous paper I [1] have demonstrated that iodized poppy-seed oil is a valuable adjunct in the operative treatment of pterygium.

1. Dimitry, T. J.: Iodized Oil Technic in Pterygium Cases, New York State J. Med. **37**: 719 (April 15) 1937.

HISTOLOGIC OBSERVATIONS

The nature of the tissue reaction following an injection of iodized poppy-seed oil is shown in figure 6. At the present time reports of histologic examination of only two specimens are available. The first specimen was a section of pterygium into which the oil had been injected and which was subsequently removed. The second was a section taken through the vitreous of a rabbit some time after the injection of the oil.

CONCLUSIONS

Iodized poppy-seed oil is a valuable opaque medium for use in obtaining roentgenograms of the eye and the periocular spaces and tissues.

The iodized oil has a definite therapeutic value in certain diseases of the eye.

No untoward reactions or toxic effects of any kind were observed in the investigation.

Dr. A. Granger, roentgenologist at the Louisiana State Hospital, assisted in this study.

A CASE OF MIXED TUMOR OF THE LACRIMAL GLAND

WITH RETINAL FOLDS AND CHOROIDAL DETACHMENT, WHICH DISAPPEARED AFTER REMOVAL OF THE GROWTH

JOSEPH ZIPORKES, M.D.
NEW YORK

So-called mixed tumor of the lacrimal gland is relatively uncommon, and to date about 275 cases of this condition have been recorded. Though the lacrimal gland is almost identical in histologic structure with the parotid and salivary glands, it appears to be much less subject to neoplastic growths than are those glands, which are situated nearer the alimentary tract. The more protected situation of the lacrimal gland as regards trauma or toxic influences suggests itself as a reason for its greater immunity.

Warthin [1] in 1901 made the first complete pathologic investigation of such a tumor. In 1905 Verhoeff [2] reported in detail 1 case of his own and 4 cases collected from the records of the Massachusetts Charitable Eye and Ear Hospital. In 1922 Lane,[3] of the Mayo Clinic, gave a short résumé of tumors of the lacrimal gland reported to that date, about 212 cases in all. Davies in 1934 described 2 cases. Dr. Arnold Knapp removed one such tumor in a case reported by Dr. Last [5] in the ARCHIVES.

Warthin was of the opinion that such tumors are of endothelial origin. However, Verhoeff, Ewing,[6] Samuels and a majority of pathol-

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This case was presented before the Section of Ophthalmology of the New York Academy of Medicine, Nov. 16, 1936.

1. Warthin, A. S.: A Case of Endothelioma of the Lacrimal Gland (Myxo-Chondro-Endothelioma Cylindromatodes) with an Analysis of Previously Reported Cases of Lacrymal Gland Tumors, Arch. Ophth. **30**:601, 1901.

2. Verhoeff, F. H.: The Mixed Tumors of the Lacrymal and Salivary Glands, J. M. Research **8**:319, 1904-1905.

3. Lane, L. A.: A Study of Tumors of the Lacrymal Gland with Report of a Mixed Tumor, Am. J. Ophth. **5**:425, 1922.

4. Davies, W. S.: Neoplasms of the Lacrimal Gland, with Report of Two Cases, Arch. Ophth. **12**:33 (July) 1934.

5. Last, Murray A.: Mixed Tumor of the Orbit of the Salivary Gland Type: Successful Removal with Preservation of Eyeball, Arch. Ophth. **13**:812 (May) 1935.

6. Ewing, James: Neoplastic Diseases: A Treatise on Tumors, ed. 2, Philadelphia, W. B. Saunders Company, 1922.

ogists have expressed the belief that they originate from congenital epithelium. The salivary and lacrimal glands are the seat of mixed tumor and are probably derived from the buccal ectoderm, with possible admixture from the maxillary periosteum or the brachial cartilage. They represent elements in the form of strands of cells, alveoli, diffuse masses of mesoblastic tissue, chiefly cartilaginous, mucous and cellular connective tissue. Any of these may predominate, giving rise to nearly pure chondroma, sarcoma or carcinoma, but usually all the types of cells are represented.

There are two points of controversy regarding this type of tumor. The first concerns its origin. It is thought that in the majority of cases this growth arises not from the lacrimal gland but from a fetal rest near the gland. The second question is whether it is truly a mixed tumor. Modern evidence indicates that the areas of mucoid, spindle cell, glandular and other types of tissue may represent a peculiar, unexplained epithelial metaplasia, and hence the term mixed tumor may be a misnomer. It may be more accurate to label the growth in the case reported here "so-called" mixed tumor of the lacrimal gland.

The clinical course of these tumors varies, but in general they are of slow growth, most likely because they usually have a distinct capsule. In the majority of cases this growth has been present long before it is brought to the attention of the ophthalmologist. The size varies, and when the tumor attains some appreciable growth it causes displacement of the eyeball. Lacrimation, as well as edema of the upper lid, is a prominent complaint. Vision decreases with the size of the growth, owing to papillitis, corneal astigmatism or even (rarely) atrophy of the optic nerve.

Most authors assert that mixed tumor of the lacrimal gland is benign. However, Lane reported 12 deaths and 19 recurrences in 95 cases of this condition. Of Verhoeff's 5 patients, 2 required exenteration of the orbit, and 1 died. Of Benedict's 5 patients, who were reported on in 1930, 2 are alive, 2 dead and 1 lost sight of. Mixed tumor shows a low grade of malignancy in the earlier stages and is important then only regionally. But if not completely removed it recurs repeatedly; its growth becomes much more accelerated, and it then definitely shows a much higher grade of malignancy. It is important to save the eyeball only if it is certain that the tumor has been completely removed. Whether a Krönlein operation or a simple incision through the lid is preferable depends on the individual case. However, one gets a bigger field with a Krönlein operation, and as it is important to remove every bit of tumor, that approach is more advisable. Some believe that irradiation should be applied to the site of removal, although Hoche and his

Fig. 1.—Cross-section of the tumor; low power magnification. The growth measured 1¼ by 1 inch (3 by 2.5 cm.) and was enclosed in a capsule 1 mm. thick.

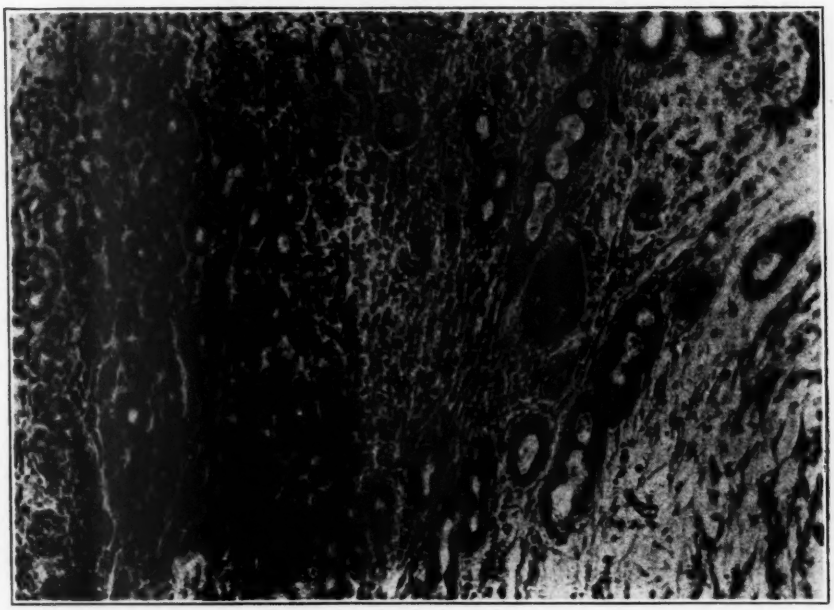

Fig. 2.—Normal lacrimal gland tissue and lacrimal duct structures at the margin of the tumor.

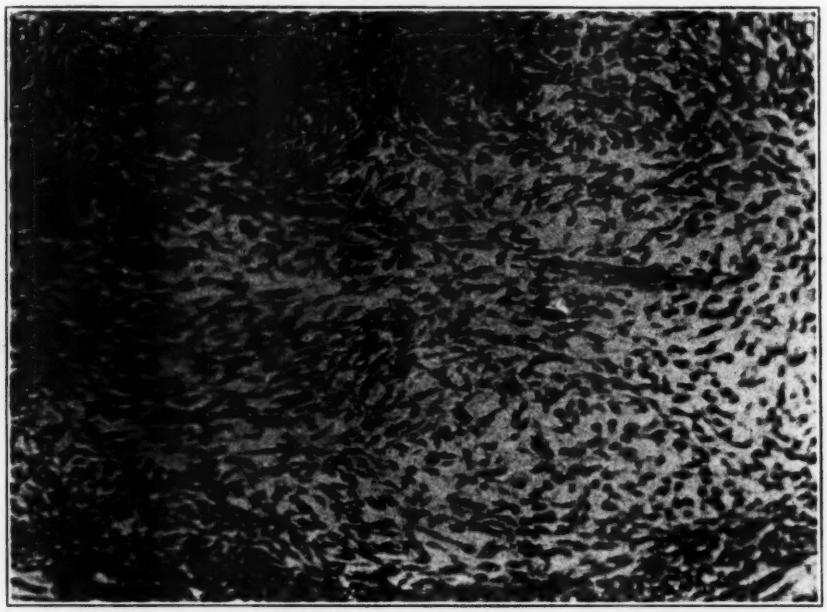

Fig. 3.—Section showing areas of myxomatous tissue and mucoid spindle cell tissue.

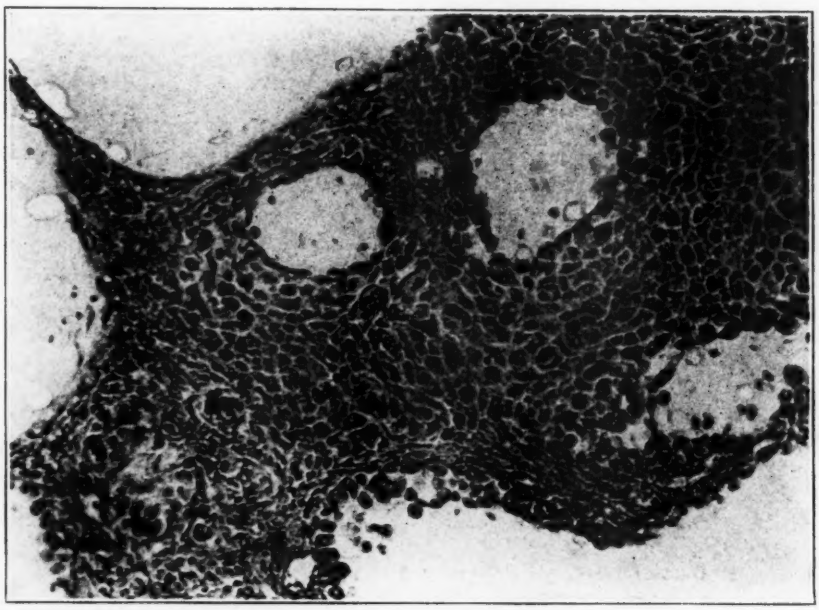

Fig. 4.—Section showing areas of pavement epithelium with cystic spaces; occasional mitotic figures; epithelial cells with fine intercellular bridges, and scattered stellate cells.

co-workers [7] stated that this tumor is absolutely refractive to radiotherapy and advise only surgical procedures.

REPORT OF CASE

A married woman 40 years of age was first seen at the Herman Knapp Memorial Eye Hospital one year ago. The complaint was ptosis in the right eye of seven months' duration. There had been tearing and blurred vision of this eye for three months. The past history revealed that twenty years previously the left submaxillary gland had been removed. The cause for this removal was unknown. Otherwise, nothing relevant was learned. Thorough general examination and all the laboratory tests gave negative results. Examination of the right eye showed vision of 20/30— and ptosis. There were limitation of motion of the eyeball up and out and diplopia in the field. The eye became slightly more prominent. There was a mass in the orbit to the inner side of the lacrimal gland, extending nasally and back. It was difficult to determine whether it was attached to the periosteum or not. The fundus presented an interesting picture: The disk was normal. There were seven or eight parallel folds in the retina, extending from the temporal side of the disk to the periphery and concentric with the disk. They resembled the white striae of retinitis often present in a detached retina which has become reattached. Where a retinal vessel passed over the folds there was a slight bend. In addition, in the extreme upper, temporal portion of the periphery there was a choroidal detachment, which was thought by some to be due to a melanosarcoma. Dr. Knapp, in a case which he reported before this section in 1929 under the title "Tumor of the Orbit Producing Retinal Folds," quoted Dimmer's statement to the effect that scleral indentations may raise the choroid in an elevation similar to that of the retina caused by a choroidal growth.

Subsequent events proved that that was the correct explanation, as after I removed the growth by a Krönlein operation in March 1936, both the folds and the detachment cleared up in about eight weeks. This growth was entirely encapsulated. Vision is now 20/20. There is no diplopia or ptosis.

The report of the pathologist, Dr. J. C. Ehrlich, was:

Grossly the tumor measured 1¼ by 1 inch (3 by 2.5 cm.). It was enclosed in a fibrous capsule 1 mm. thick. Histologically there were the following distinct features:

1. Normal lacrimal gland tissue and lacrimal duct structures at the margin of the tumor, outside of the capsule.

2. Areas of myxomatous tissue and mucoid spindle cell tissue.

3. Areas of pavement epithelium with cystic spaces.

4. Occasional mitotic figures in the nucleus of some cells.

5. Epithelial cells with fine intercellular bridges (perhaps the so-called prickle cells).

6. Scattered stellate cells, which may have been precartilaginous cells.

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7. Hoche, Baudot, Gault and Legait: Tumeur mixte de la glande lacrymale, Bull. Soc. d'opht. de Paris, April 1935, p. 249.

EUGENIC SIGNIFICANCE OF RETINITIS PIGMENTOSA

WILLIAM ALLAN, M.D.
CHARLOTTE, N. C.

Retinitis pigmentosa may be defined briefly as a degeneration of the retina in which "there are atrophy and destruction of the retinal neuro-epithelium, the rods and cones, and their cell bodies" [1] "with hyperplasia of connective tissue and advancement of pigment cells or their granules into the vacant retinal spaces." [2] The natural consequence of this lesion is poor vision, which is aggravated, of course, by poor light.

Retinitis pigmentosa may be inherited as a dominant, a recessive or a sex-linked recessive trait, and this diversity in hereditary pattern is doubtless the reason why the subject is so confused in the medical literature, which shamelessly reveals the unfamiliarity of ophthalmologists with the subject of genetics. Wilmer,[2] for instance, agreed with Nettleship that the ratio of males affected to females affected is 3:2; both dominant and recessive heredity, of course, affect the sexes equally, but in cases of sex-linked recessive traits only the males are affected, and this is doubtless the reason for the preponderance of males with this condition. Wilmer quoted Nettleship to the effect that only thirty-six fathers will transmit the trait to their offspring for every fifty mothers that do likewise. The two sexes transmit dominant traits equally, and parents transmit recessive traits jointly, but in the case of sex-linked recessive traits the affected males transmit only through their daughters, and this is doubtless the explanation for the preponderance of female transmitters. Although different persons even in the same family vary somewhat in the amount of degeneration they inherit to start with, and although nothing is known about the effect of environment on the progress of the lesion, yet the pattern of inheritance is apparently the chief factor in determining the degree of loss of vision and the age when this begins.

Read at the Fifty-Second Annual Meeting of the Association of American Physicians, Atlantic City, N. J., May 5, 1937, and at the Twenty-Fifth Annual Meeting of the Eugenics Research Association, New York, June 5, 1937.

1. Friedenwald, J. S., and Chan, E.: Pathogenesis of Retinitis Pigmentosa. Arch. Ophth. **8**:172 (Aug.) 1932.

2. Wilmer, W. H.: Hereditary Factors Responsible for Development of Optic Atrophy and Retinitis Pigmentosa, Arch. Neurol. & Psychiat. **12**:136 (Aug.) 1924.

The first two pedigrees show the inheritance of retinitis pigmentosa as a dominant trait. Those afflicted usually become aware of night blindness between the ages of 25 and 50, sometimes earlier, and the degeneration gradually progresses. But of the thirty-eight persons affected in the two families, only one became too blind to work, and that after 50.

It will be seen in these two pedigrees that retinitis pigmentosa passes down through and affects the sexes equally and that every one affected got it from one parent and passed it on to half the children. Thus it has been inherited as a unitary dominant trait in these two families.

The first three generations of the first family grew up in the era of candle light, and the five women ranging in age from 77 to 97 who gave me the history were very definite about who was night blind or

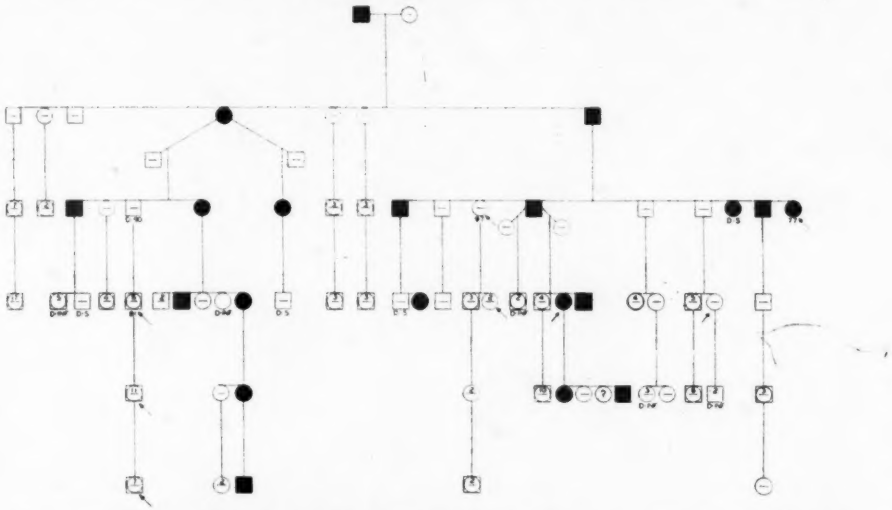

Fig. 1.—Pedigree showing inheritance of retinitis pigmentosa as a dominant trait. In all the pedigrees the squares indicate males and the circles females. *D* indicates died; *D—INF* indicates died in infancy, and *S* indicates single. The squares and circles with a horizontal line show those unaffected, and the black squares and circles show those affected. The numbers below the symbols are the ages in years. The numbers inside the squares, circles and circles within squares indicate the number of children, of both sexes. The arrows indicate members actually seen or examined.

moon eyed in the family and who was not. Reports of ophthalmoscopic examination on the younger generations were secured (members 5, 18, 21 and 26 of the fourth generation, members 3, 6, 7, 8 and 9 of the fifth generation and member 3 of the sixth generation).

The second family had used electric light most of the time, and as many of the members were uncertain whether they were night blind or not it was necessary to carry an ophthalmoscope over two counties and examine them.

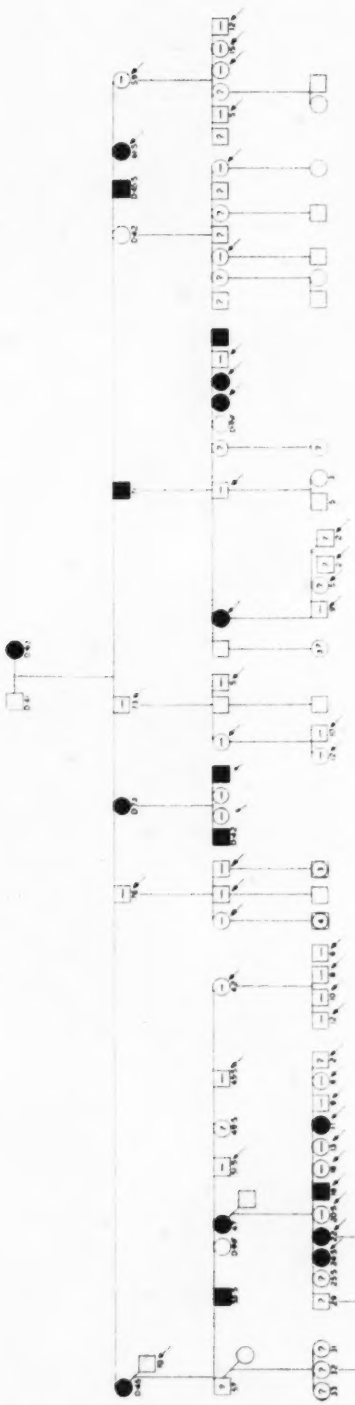

Fig. 2.—Pedigree showing inheritance of retinitis pigmentosa as a dominant trait. The question marks inside the squares or circles indicate that it was not known whether these members were affected.

In the following pedigrees (figs. 3, 4, 5, 6, 7 and 8), which show that retinitis pigmentosa has been inherited as a recessive trait, the affected members became very blind before the age of 25 years. In many pedigrees which show the inheritance of retinitis pigmentosa as a recessive trait the kinship of the parents was close enough and recent enough to be within the knowledge of the descendants, as in the pedigrees shown in figures 3, 4, 5 and 6; in others, kinship of the parents was too remote to be within the knowledge of the descendants, or the marriage brought together members of two unrelated families in both of which retinitis was carried as a recessive trait.

In the family the pedigree of which is shown in figure 3, I examined with the ophthalmoscope the parents, ten of the eleven children and

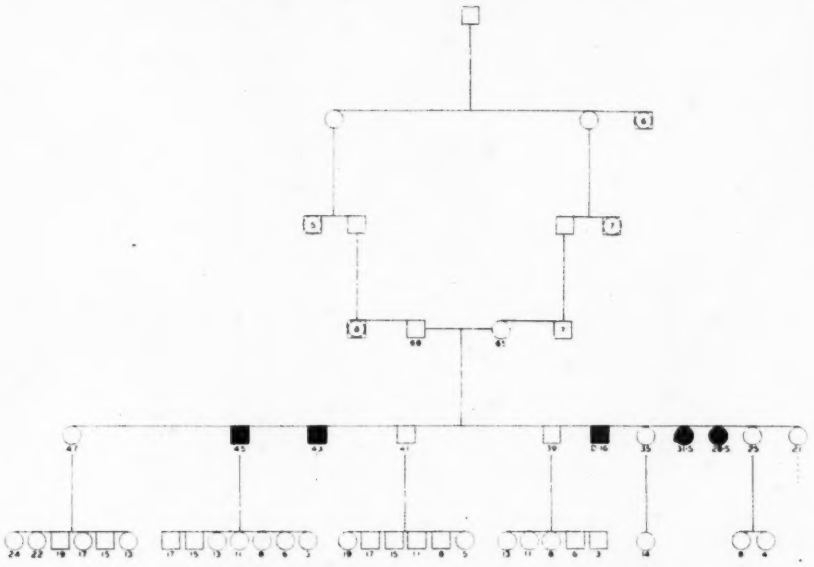

Fig. 3.—Pedigree showing inheritance of retinitis pigmentosa as a receessive trait.

some twenty-five grandchildren. Members over 80 on both sides of the family remembered only one of the kin who had ever gone blind—a man who lost his sight after the age of 75, doubtless from cataract. A woman of 83 years had heard her mother speak of two men, a father and son, who had the same surname as members of the first generation; the father was called "See" Ben and his son "Blind" Ben. "Blind" Ben had been kicked by a horse at the age of 17, but as this was about one hundred and fifty years ago it was not known whether this caused the blindness or exactly what kin these men were to member 1 of the first generation. It seems evident that in this family retinitis pigmentosa was a recessive trait brought into evidence by the marriage of cousins in the third generation.

The first of the two women whose pedigree is shown in figure 4 dated her severe blindness from an attack of malaria at the age of 22; the second sought treatment for poor vision at about the same age. Their parents were first cousins on one side of the family and second cousins on the other.

When the men whose pedigree is shown in figure 5 were choir-boys they had to be led to choir practice on Friday night; their parents were first cousins.

Two of the afflicted members of the family the pedigree of which is shown in figure 6 were seen in a county home, the other two having died

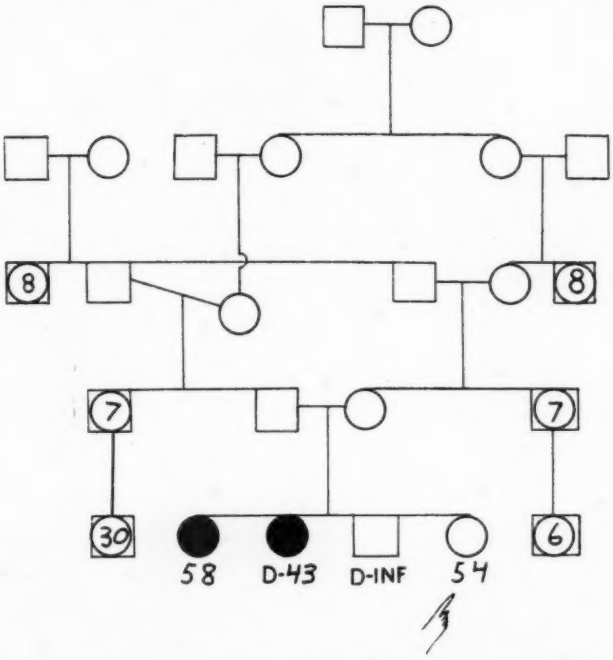

Fig. 4.—Pedigree showing inheritance of retinitis pigmentosa as a recessive trait. The pointing hand indicates a member actually seen or examined.

in county homes. They were too old and garrulous to give any concise statement as to just when they lost useful vision. Their mother was the niece of their father.

The two afflicted members of the family the pedigree of which is shown in figure 7 had been very blind for some years. I examined both parents, who knew little about their grandparents' generation but were not cognizant of any intermarriage or blindness in the family.

The two members with retinitis pigmentosa of the family the pedigree of which is shown in figure 8 became very blind in the second decade of life. I examined the father, who knew of no intermarriage or blindness in the family.

The reason for the difference in the severity of retinitis pigmentosa according to its mode of inheritance seems obvious. When an uncommon condition is inherited as a unit dominant trait, those affected will all be heterozygous for the trait; that is, the trait will be conditioned by a single defective autosomal gene, derived from one parent. But when such a trait is recessive, all those showing it will be homozygous for it; that is, the trait will be conditioned by both members of the pair of defective genes, one derived from each parent.

If this is the proper explanation of the much greater severity and earlier appearance of blindness when retinitis is inherited as a simple

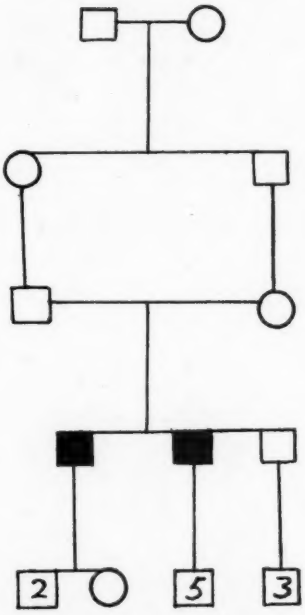

Fig. 5.—Pedigree showing inheritance of retinitis pigmentosa as a recessive trait.

recessive trait, its behavior when inherited as a sex-linked recessive trait is a matter of considerable interest. Will the defective gene on the single x-chromosome of the male behave like the single defective gene, balanced by a normal gene, in dominant heredity, and produce only half blindness or night blindness; will it behave like the pair of defective genes, without any counter-balancing normal genes, in recessive heredity and produce early and severe blindness, or will it behave in an intermediate manner because there is, on the one hand, no normal gene to modify its action and, on the other, no second defective gene to intensify its action? The pedigree shown in figure 9, in which retinitis pigmentosa has been inherited as a sex-linked recessive trait, suggests

that this type of inheritance occupies a place midway between dominant and recessive inheritance. The two affected brothers in the fifth generation, who are still living, noticed impaired vision in the second decade

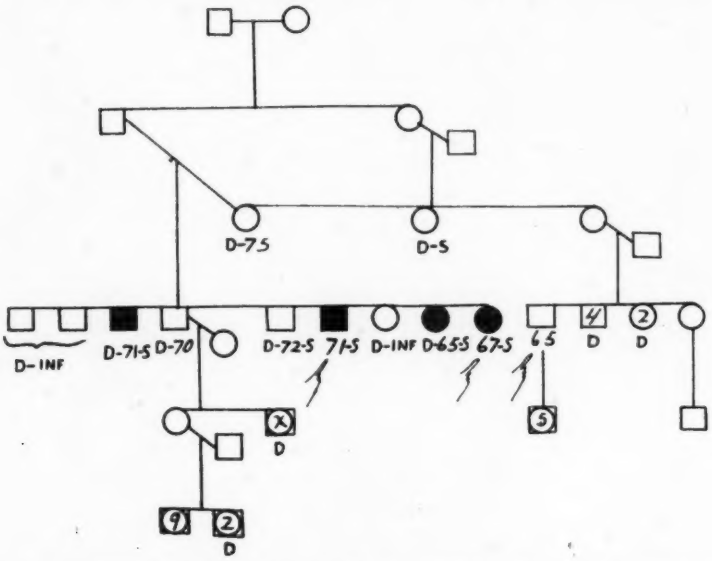

Fig. 6.—Pedigree showing inheritance of retinitis pigmentosa as a recessive trait. The pointing hands indicate members actually seen or examined. The X within the circle in the square indicates that the number of sons was unknown.

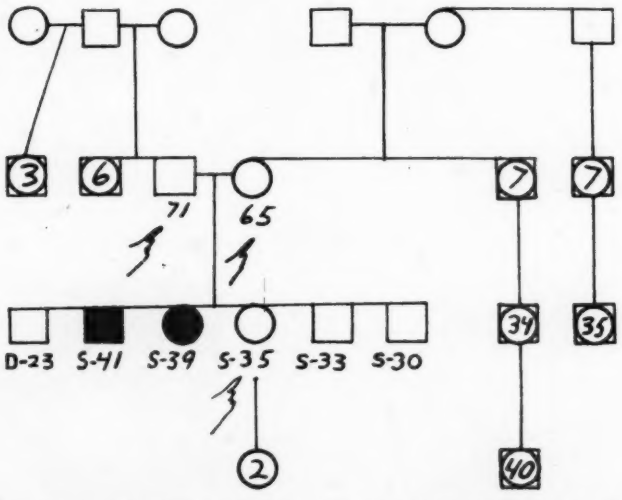

Fig. 7.—Pedigree showing inheritance of retinitis pigmentosa as a recessive trait. The pointing hands indicate members actually seen or examined.

and almost complete blindness by the time they were 45 years old. The brother who died at 21 was noticeably night blind, as is the nephrew at 19.

This family illustrates some of the difficulties in dealing with human pedigrees. The mother had only two sibs, a brother who was not blind and a sister who was not a carrier. The mother's mother and all her sibs died early of tuberculosis, so there were no collateral descendants from this generation. The mother's maternal grandmother, who was

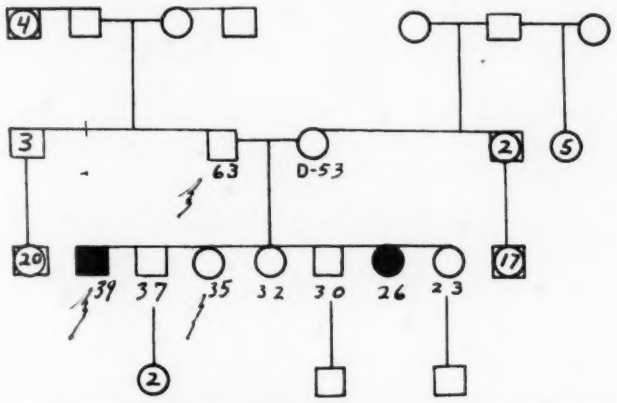

Fig. 8.—Pedigree showing inheritance of retinitis pigmentosa as a recessive trait. The pointing hands indicate members actually seen or examined.

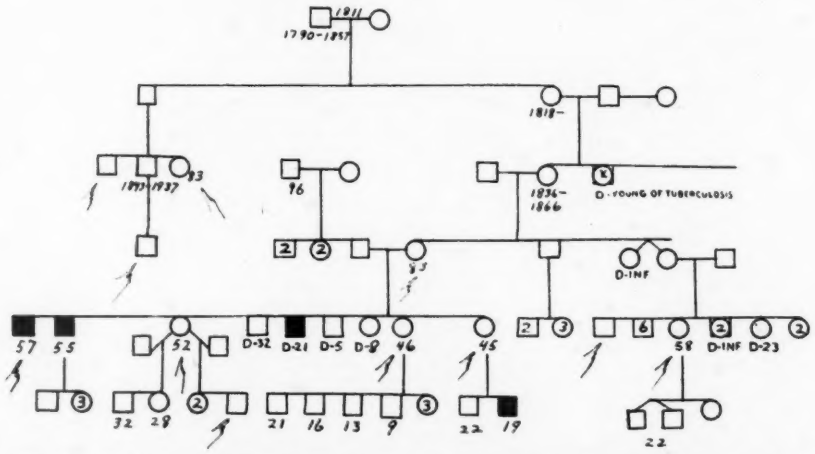

Fig. 9.—Pedigree showing inheritance of retinitis pigmentosa as a sex-linked recessive trait. The pointing hands indicate members actually seen or examined. The X within the circle in the square indicates that the number of sons was unknown.

born in 1818, had sibs, and several descendants of these sibs have been interviewed, but no trace of retinitis pigmentosa was found.

Dr. Wilmer advised that if a child with this lesion is born there should be no more children in that family.

There are various reasons why this advice is not practical. 1. In cases of recessive heredity the number of carriers is twice the number of those who show the trait. 2. In cases of dominant heredity, as regards retinitis pigmentosa, the degeneration may not cause symptoms for twenty or thirty years; member 4 of the third generation of the family the pedigree of which is shown in figure 2, for instance, knew nothing of night blindness in her family until one of her younger girls was big enough to fall over a wheelbarrow one night when going to the barn to milk—and there were already twelve children by that time. 3. Even in the family the pedigree of which is shown in figure 3 it was not until the older children were well along in school that blindness was discovered, and by that time there were already seven or eight children in this family. 4. Ophthalmoscopic examination is not available in the deep country. 5. Examination of children too young to cooperate is very unsatisfactory. 6. In the cases of dominant heredity at least, pigment is probably not present in the early years.

Vision, roughly speaking, depends on two factors—sound eyes and good illumination. Vision will vary directly as both factors are good or bad, but if either factor alone is poor it may be compensated for by excellence in the other factor, and satisfactory vision still result.

In the era of candle light, which lasted until the era of the coal oil lamp, night blindness must have been a severe handicap. In the present era of the electric light, in which electric light is available even in the deep country, the intensity of ordinary household illumination has increased from seventy-five-fold to a hundredfold, and this improvement in light has largely compensated for the defect in eyes heterozygous for retinitis pigmentosa, permitting satisfactory vision in the large majority of cases. Hence good illumination rather than birth control seems necessary when this lesion is inherited as a dominant trait.

When it is inherited as a simple recessive trait, avoidance of marriages of cousins in families which carry the trait becomes imperative, as shown by the pedigrees in figures 3, 4, 5 and 6. The present difficulty with this situation is that there are no family records to show in what families retinitis pigmentosa is carried as a recessive trait. Obviously such records should be started now by the public health authorities in each county, and educational work should be done to prevent hereditary blindness; laws are not necessary to keep women from having blind babies. In the pedigree shown in figure 3, for instance, each of the fifteen sibs of the parents of the blind children has or had one chance in two of carrying the trait, and each of the eighty-four children of these fifteen sibs—the first cousins of the blind children—has had one chance in four of carrying the trait. Each of the six sibs of the blind children has one chance in two of carrying the trait, and each of their twenty children has one chance in

four. The seven children of the blind man will, of course, all be carriers of retinitis pigmentosa, and their children, if any, will have one chance in two of being carriers. Member 1 of the sixth generation in this pedigree married her half first cousin once removed, and another member of this clan married the daughter of member 4 of the fourth generation in pedigree 6, who lived in a nearby district; by the law of chance none of the seventeen children resulting from these two unions are blind, but only ignorance permits the taking of such chances.

When retinitis pigmentosa is inherited as a sex-linked recessive trait, half the sisters and all the daughters of any afflicted man will pass the trait to half their sons. In this situation the responsibility rests largely on the women in the family, and I find from experience in applying negative eugenics that a warning to the women in these families that the chances are 100, 50 or 25 per cent that some of their boys will be blind stops reproduction. Those who are kin to the blind know the weight of this cross.

From the foregoing remarks it is evident that ignorance of the laws of genetics is the prime factor in the continued production of the severer types of retinitis pigmentosa—those seen in instances in which the condition is inherited as a recessive trait or as a sex-linked recessive trait. Plainly, it is the duty of the medical profession to advise members of families with such heredity.

Shall physicians embrace this opportunity to practice preventive medicine by making knowledge of genetics as necessary a part of medical training as knowledge of bacteriology, or shall the blind be allowed to continue to lead the blind?

IMPORTANCE OF DIET IN THE ETIOLOGY AND TREATMENT OF TOBACCO-ALCOHOL AMBLYOPIA

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It is my purpose to present the results of a clinical investigation which I think is unusual and, judged by older ideas, quite unorthodox. For more than two years patients with tobacco-alcohol amblyopia have been allowed to continue their usual intake of tobacco and alcohol provided they consumed the diet prescribed for them. This work has been carried out slowly and cautiously. All the patients have been hospitalized throughout the period that they were permitted to smoke and drink. The course has been carefully followed by frequent examinations of the visual fields and visual acuity. The daily intake not only of tobacco and alcohol but of every component of their diet over this entire period of hospitalization is accurately known.

First, the previous studies, not made by ophthalmologists, which have a bearing on this subject will be considered. Nine years ago the suggestion was made [1] that vitamin deficiency may be an important factor in the etiology of alcoholic polyneuritis. The following year this suggestion was repeated,[2] and then came reports of cases of alcoholic polyneuritis in which the patient was greatly benefited by the proper diet.[3] In 1931 an internist [4] mentioned that a number of cases of retrobulbar neuritis due to vitamin B deficiency had occurred in Japan, and he expressed the opinion that retrobulbar neuritis occurring in patients with chronic alcoholism should be investigated from this standpoint. In 1933 Spies and De Wolf [5] allowed ten hospitalized patients with the alcoholic type of pellagra to drink between 1 pint (475 cc.) and 1 quart

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From the Institute of Ophthalmology, the Columbia Presbyterian Medical Center.

Read before the Section on Ophthalmology at the Eighty-Eighth Annual Session of the American Medical Association, Atlantic City, N. J., June 11, 1937.

1. Shattuck, G. C.: Am. J. Trop. Med. **8**:539, 1928.

2. Minot, G. R.: Ann. Int. Med. **3**:216, 1929.

3. Wechsler, I. S.: M. J. & Rec. **131**:441, 1930; Etiology of Polyneuritis, Arch. Neurol. & Psychiat. **29**:813 (April) 1933.

4. Keefer, C. S.: New England J. Med. **205**:1086, 1931.

5. Spies, T. D., and De Wolf, H. F.: Am. J. M. Sc. **186**:512, 1933.

(945 cc.) of whisky daily, provided they took a well balanced diet plus 75 Gm. of yeast daily. The patients recovered from the pellagra well, despite the large intake of alcohol.

Early in 1935, after a previous study of fifty-five cases [6] of tobacco-alcohol amblyopia and after consideration of the known effects or, better, the lack of effect of alcohol on the nervous system, the present investigation was begun. While the first patient was under treatment a report [7] was published from the Boston City Hospital stating that ten patients with alcoholic polyneuritis had been hospitalized, allowed to consume their usual intake of alcohol, which was between 1 pint and 1 quart of whisky daily, and fed a well balanced, high vitamin diet supplemented with yeast and other vitamin B-containing substances. This was a procedure similar to what I had started with the patients having tobacco-alcohol amblyopia, and it was encouraging to learn that the polyneuritis had improved in every instance and that the rapidity and degree of recovery were apparently just the same in patients who were allowed to drink this large quantity of alcohol as in patients who were forced to abstain from alcohol. In 1934 an investigator [8] in physiologic chemistry calculated what seemed to be the vitamin B requirement of man. Using these calculations, physicians at the Bellevue Hospital [9] in New York have found that every alcohol addict with polyneuritis has had an inadequate intake of vitamin B. There has accumulated a considerable number of reports all tending to indicate the same thing, i. e., that so-called alcoholic polyneuritis is a manifestation of a deficiency disease and is not due to a direct neurotoxic effect of the alcohol.

The present investigation will now be considered. Patients with tobacco-alcohol amblyopia were informed of the nature of their disease and told of the usual treatment for this type of amblyopia, but they were also told that on certain conditions they could enter the hospital without charge to them. The conditions were: (1) that they should continue to smoke and drink as much as they had while contracting the disease, and (2) that they must eat what was given them.

Eight patients have been studied in this way. I shall report on each of them separately. At this time I should like to present the report of one case (case 8) which illustrates the method used and the therapy employed.

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6. Carroll, F. D.: Analysis of Fifty-Five Cases of Tobacco-Alcohol Amblyopia, Arch. Ophth. **14**:421 (Sept.) 1935.

7. Straus, M. B.: Am. J. M. Sc. **189**:378, 1935.

8. Cowgill, G. R.: The Vitamin B Requirement of Man, New Haven, Conn., Yale University Press, 1934.

9. Jolliffe, N.; Colbert, C. N., and Joffe, P. M.: Am. J. M. Sc. **191**:515. 1936.

REPORT OF A CASE

CASE 8.—W. H., a 39 year old Negro, was seen by me on Nov. 8, 1935, at the Vanderbilt Clinic. He stated that his vision had been gradually decreasing for seven weeks. He drank between 1 pint and 1 quart of cheap corn whisky daily and smoked one and a half packages of cigarets daily. Vision was 20/200 in each eye, and the fields showed centrocecal scotomas, which are always present in the eyes of persons with tobacco-alcohol amblyopia; the rest of the ocular examination gave negative results. One week later, vision had decreased to 15/200 in the right eye and to 10/200 in the left eye (fig. 1 *A*), and the patient was admitted to the Institute of Ophthalmology. Gastric analysis revealed absence of free hydrochloric acid. Throughout the period of hospitalization he smoked his usual number of cigarets daily and was given between 1 pint and 1 quart of liquor daily. I purchased a quantity of the corn whisky he had been drinking, had it examined chemically and then gave it to him for consumption in the hospital. Chemical analysis failed to show any appreciable quantity of any toxic substance except ordinary ethyl alcohol, and this has been the chemical finding in numerous other samples of liquor used by other patients with this type of amblyopia. The diet which he was given was a high vitamin B, well balanced diet adequate in all respects and supplemented by powdered brewers' yeast [9a] in doses of 2 tablespoonfuls five times daily; vegex (a brewers' yeast extract) in doses of 1 teaspoonful three times daily and wheat germ (Embo) in doses of 4 tablespoonfuls three times daily. He was also given liver extract [9b] intramuscularly in doses of 5 cc. several times weekly and cod liver oil in doses of 1 ounce (30 cc.) daily. This therapy will be referred to later merely as the "prescribed diet."

While the patient was on this prescribed diet and while he was having his usual intake of tobacco and alcohol his vision gradually improved in seven weeks from 15/200 in the right eye and 10/200 in the left eye to 20/25 and 20/30+, respectively. The fields of vision showed a corresponding improvement, and he was then discharged from the hospital. He was advised to decrease his consumption of alcohol because of any possible effect that such large quantities of alcohol might have on other organs of the body, but he has not followed this advice very well. He was also advised to take 2 tablespoonfuls of powdered brewers' yeast daily, and he has followed this advice somewhat better. Seventeen months have passed since his discharge from the hospital. Vision is 20/20 in each eye (fig. 1 *B*).

COMMENT

All these patients have been followed at least one year. One of the eight patients did not improve. She had atrophy of the papillomacular bundle of the optic nerve, and vision remained stationary. All the other patients showed very satisfactory results, and all have vision for reading. The speed of recovery seemed to be at least as good in these patients as in patients previously studied who abstained from tobacco and alcohol. But I cannot say that this will prove to be true in a larger series of patients. The course of this disease is variable. It has been known that patients may sometimes improve considerably who have merely decreased somewhat their intake of alcohol or tobacco, and infrequently a patient who continued to smoke and drink as much as before

9a. Budweiser yeast was used.

9b. Solution liver extract-Lilly N. N. R. was employed.

the development of the amblyopia has been known to experience a return to normal vision. But this is most unusual. Certainly, on the basis of previous knowledge of this condition such marked improvement in seven of eight patients who failed to decrease their intake of alcohol or tobacco would never be expected.

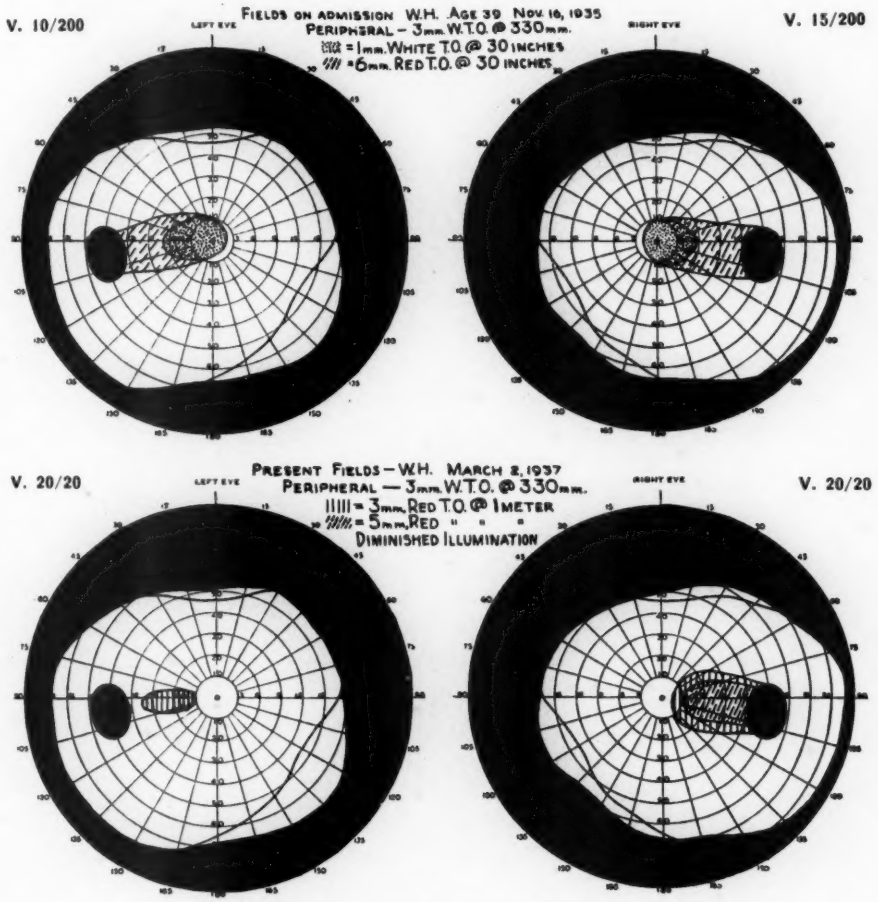
V. 10/200
FIELDS ON ADMISSION W.H. AGE 39 NOV. 16, 1935
PERIPHERAL — 3mm. W.T.O. @ 330mm.
= 1mm. WHITE T.O. @ 30 INCHES
= 6mm. RED T.O. @ 30 INCHES
V. 15/200
V. 20/20
PRESENT FIELDS — W.H. MARCH 2, 1937
PERIPHERAL — 3mm. W.T.O. @ 330mm.
= 3mm. RED T.O. @ 1 METER
= 5mm. RED " " "
DIMINISHED ILLUMINATION
V. 20/20

Fig. 1 (case 8).—Fields of W. H.

That the general nutrition of the patient plays a role in the development of this disease is not a new idea.[10] Further, this type of amblyopia

10. Mackenzie, William: A Practical Treatise on the Diseases of the Eye, Philadelphia, Blanchard & Lea, 1855, p. 987. Traquair, H. M.: Edinburgh M. J. **42**:153 (Aug.) 1935. Holth, S.: Acta ophth. **5**:195, 1927. Letchworth, T. W.: Clin. J. **61**:137, 1932. Yudkin, A. M.: Ocular Disturbances Produced in Experimental Animals by Dietary Changes: Clinical Implications, J. A. M. A. **101**:921 (Sept. 16) 1933; Vitamins: Clinical Evidence of Necessity of Vitamins for Function of Ocular Tissue, Arch. Ophth. **14**:112 (July) 1935. Carroll.[6]

is found [11] in patients with alcoholic polyneuritis and in those with the alcoholic type of pellagra, which are almost surely deficiency diseases. In addition, a type of amblyopia occurs in persons with beriberi [12] which is said to be indistinguishable by ocular findings from tobacco-alcohol amblyopia.[13]

Is it justifiable, then, on the basis of all the facts now known, to state at this time that the condition is purely the result of a deficiency state? I do not think so. If that were true, one might reasonably expect to see some other manifestation of deficiency disease in these patients. By the present tests one does not find that in most of these patients. And if that were true, one might also reasonably expect to find a type of amblyopia indistinguishable from tobacco-alcohol amblyopia among patients who do not smoke or drink but who have deficiency conditions such as pellagra. And as has just been stated, there is a possibility that some patients with beriberi do have amblyopia of this type, but at the present time I do not think that there is adequate evidence on this point. Through the agency of numerous physicians in the southern states I recently had the opportunity of examining the eyes of one hundred and ten patients with pellagra who did not smoke or drink in appreciable amounts. None of these patients with pellagra showed any evidence of toxic amblyopia, but numerous patients with pellagra in New York have alcohol amblyopia, and they are all heavy drinkers.

What, then, is the explanation of the production of tobacco-alcohol amblyopia? Perhaps no one can yet give the full answer to that question. Experimentation [14] has shown that animals in a poor state of nutrition are much more susceptible to the injurious action of poisons and toxins than other animals, and it is not unlikely that in the production of this type of amblyopia patients suffering from subclinical nutritional deficiency have a markedly increased susceptibility to tobacco and alcohol. But this is pure theory, and I shall change my theory whenever new facts indicate such change. It is no theory that a series of these

11. Carroll, F. D.: "Alcohol" Amblyopia, Pellagra, Polyneuritis; Report of Ten Cases, Arch. Ophth. **16**:919 (Dec.) 1936.

12. Elliot, R. H.: Tropical Ophthalmology, London, Oxford University Press, 1920, p. 407.

13. Komoto, Jufiro (Tokio, Japan): Personal communication to the author.

14. Rose, S. B.; Rose, W. B., and Kolmer, J. A.: J. Infect. Dis. **59**:50, 1936. Werkman, C. H.; Baldwin, F. M., and Nelson, V. E.: ibid. **35**:549, 1924. Nedzel, A. J.: J. Lab. & Clin. Med. **20**:944, 1935. Wien, R.: Quart. J. Pharm. & Pharmacol. **9**:48 (Jan.-March) 1936. Cutler, J. T.: J. Pharmacol. & Exper. Therap. **45**:209, 1932.

patients have been given an excellent diet supplemented by certain vitamin products and allowed to continue their usual intake of tobacco and alcohol and that the results have been very satisfactory.

I wish to state that I have not recommended continuing the use of tobacco and alcohol in the ordinary case. The patients in this series have all been hospitalized and observed closely over a long period. There are other patients who have refused to abstain from alcohol and tobacco and who have taken the prescribed quantity of yeast at home. Thus far they have made satisfactory improvement, but it is too early to more than mention this group. This problem is still in the stage of clinical research, but I have no hesitancy in concluding that in the etiology and treatment of this condition diet does play an important role.

REPORT OF THE OTHER CASES

CASE 1.—C. S., a 28 year old Negro woman, who was mentioned previously,[15] was first examined in the Vanderbilt Clinic in October 1934. She had marked tremor of her tongue and fingers, and her breath had a strong alcoholic odor. The vision was 20/200 in each eye and was unimproved with glasses. The disks showed very marked temporal pallor consistent with atrophy of the papillomacular bundle, and the fields revealed depression of central vision. She had never used tobacco in any form but consumed between a pint and a quart of whisky daily. In three months vision improved to 20/70 in each eye and remained stationary. She was strongly advised to discontinue the use of alcohol, but she refused. Nine months after coming under observation she was admitted to the Institute of Ophthalmology. The disks appeared atrophic temporally. She had alcoholic gastritis, and two gastric analyses showed absence of free hydrochloric acid. During thirty-six days in the hospital she drank between 1 pint and 1 quart of liquor daily. (The liquor given to her and to all other patients and referred to hereafter merely as "liquor" consisted of equal parts of 95 per cent ethyl alcohol and water flavored with the juices from lemon and orange peels. It had a pleasant odor and taste.) She took the prescribed diet. After this period she felt that her vision had definitely improved. I was certain that her eyes were not worse. She could read several lines lower on the test chart than on admission, but I felt that her condition should be considered unimproved. She has been followed twenty-two months since discharge from the hospital, and her eyes have shown no further change. The diagnosis was alcoholic amblyopia and partial optic atrophy, unimproved.

CASE 2.—G. J., a 31 year old actor, a private patient of Dr. Gordon Bruce, entered the Institute of Ophthalmology on Dec. 3, 1935. He drank about 1 quart of gin and smoked from one to two packages of cigarets daily. Vision was 20/200 in each eye. Each disk was orange except temporally, where it was somewhat pale. The margins were indistinct, except temporally, and slightly elevated (less than 1 D.). Elevation of the disk is unusual in this condition. The fields showed typical centrocecal scotomas. Neurologic examination, roentgenograms of the head and the Wassermann test gave negative results. A medical consultant

15. Carroll, F. D., and Franklin, C. R.: Am. J. Ophth. **19**:1070, 1936.

diagnosed alcoholic gastritis and hepatitis. In the hospital the patient took from 1 pint to 1 quart of liquor daily, smoked from one to two packages of cigarets daily and faithfully ate what was given to him. In two weeks vision was 20/120 in the right eye and 20/50 in the left, and the margins of the disk appeared less elevated. In one month vision was 20/40 in the right eye and 20/30 in the left, and the scotomas had greatly decreased in size. The patient was discharged from the hospital on Jan. 24, 1936, and three months later vision was 20/30 in the right eye and 20/20 in the left. He has not abstained from alcohol or tobacco, but he stated in a recent communication that his vision has remained the same.

Case 3.—N. M.,[11] a 36 year old janitor, drank about 1 quart of liquor and smoked one package of cigarets daily. He was first seen in the Bellevue Psychiatric Hospital on Dec. 12, 1935. He had pellagra, polyneuritis and alcohol amblyopia. Vision was 10/200 in the right eye and 8/200 in the left. Each disk showed marked temporal pallor compatible with optic atrophy, and the fields had centrocecal scotomas. When his condition permitted he was transferred to the Institute of Ophthalmology. Retinoscopic examination indicated a large astigmatic error in the left eye, and he stated that vision had never been good in that eye. For the first month in the hospital he was forced to abstain from all alcohol and tobacco, and there was slight improvement. Visual acuity increased from 18/200 in the right eye and 12/200 in the left eye to 20/120 in each eye with correction. He was then given from 1 pint to 1 quart of liquor daily and cigarets. He faithfully took his diet. In one month on this regimen vision increased to 20/50 in the right eye and to 20/120 in the left and the fields were improved, and in another five weeks, when he was discharged, vision was 20/40 in the right eye and 20/80 in the left.

Case 4.—W. F., a 49 year old Jewish waiter, had noticed decreased vision for four months. He had been to two ophthalmic clinics and when first seen by me was at the Neurological Institute, where he had been sent because of a suspected tumor of the brain. He smoked from ten to thirteen cigars daily and drank an average of two or three glasses of beer and an ounce (30 cc.) of whisky daily. Vision was 20/200 in each eye; the disks were normal in appearance, and the fields showed characteristic centrocecal scotomas (fig. 2 *A*). He was transferred to the Institute of Ophthalmology on May 13, 1936, and placed on the prescribed diet. He continued to smoke from nine to fifteen cigars daily and was given 6 ounces (177 cc.) of liquor daily. Neurologic examination showed some increase in the deep reflexes on the left. An otologic check-up revealed early toxic neuritis of both eighth nerves, and the audiograms were similar to those previously reported.[16] The diameter of the right pupil was 1 mm. larger than that of the left; there were small opacities in both the cornea and the lens of each eye. In two months vision was 20/50 — in the right eye and 20/60 in the left. In the next two months visual acuity did not improve, but the patient reduced his consumption of alcohol and tobacco somewhat. In three and a half months vision was 20/30 in each eye and the fields were as shown in fig. 2 *B*.

Case 5.—E. F., a 32 year old Negro postoffice clerk, who was previously mentioned,[11] had the alcoholic type of pellagra, polyneuritis and amblyopia. When

16. Carroll, F. D., and Ireland, P. E.: Association of Toxic Deafness with Toxic Amblyopia Due to Tobacco and Alcohol, Arch. Otolaryng. **21**:459 (April) 1935.

he entered the Institute of Ophthalmology on May 14, 1935, vision was 3/200 in each eye (fig. 3*A*). He abstained from alcohol and took all the prescribed food for thirty-five days. Vision by then had improved to 20/40 + in each eye, and he was discharged. He immediately started to drink and smoke as much as previously but regularly took the powdered brewers' yeast and wheat germ as prescribed. Vision gradually improved to 20/20 in each eye, and the fields showed

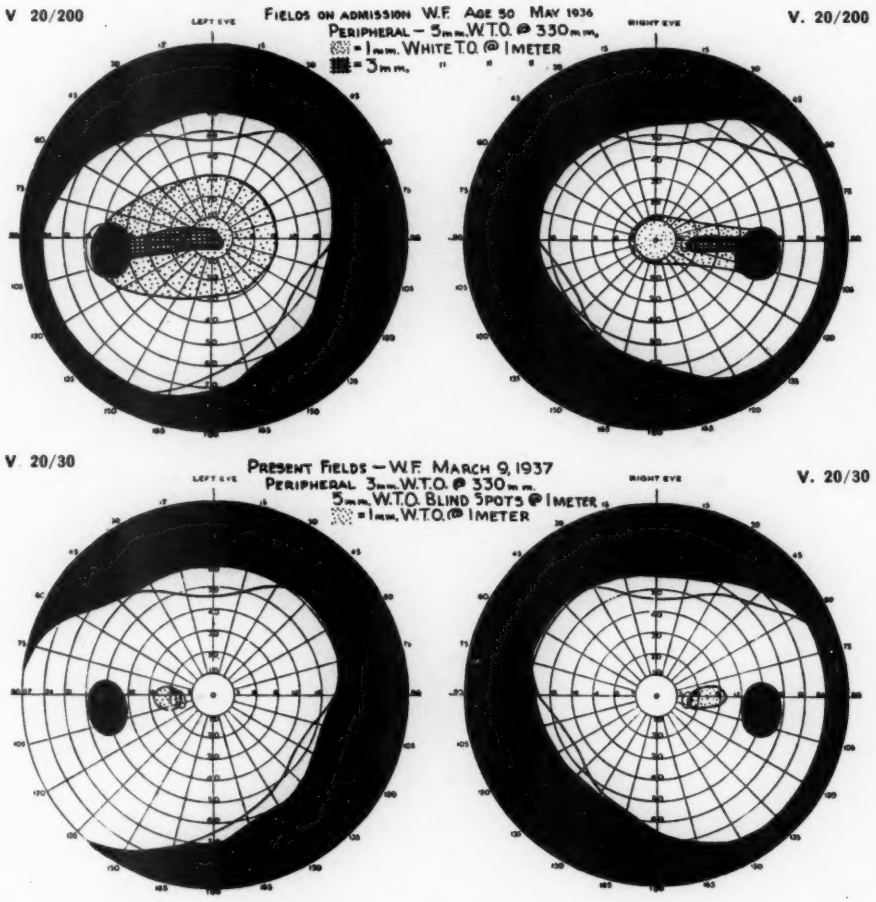
V 20/200
FIELDS ON ADMISSION W.F. AGE 50 MAY 1936
PERIPHERAL – 5mm. W.T.O. @ 330mm.
= 1mm. WHITE T.O. @ 1 METER
= 3mm. " " "
V. 20/200
V 20/30
PRESENT FIELDS – W.F. MARCH 9, 1937
PERIPHERAL 3mm. W.T.O. @ 330mm.
5mm. W.T.O. BLIND SPOTS @ 1 METER
= 1mm. W.T.O. @ 1 METER
V. 20/30

Fig. 2 (case 4).—Fields of W. F.

only a small indefinite scotoma between the blindspot and the fixation point. Then he stopped taking yeast but continued his usual intake of whisky (from 1 pint to 1 quart daily). In about five weeks the pellagra returned, and he came back to the clinic. He had mild typical pellagral dermatitis, pain in his legs and feet and diarrhea. His vision was still excellent. He was given more yeast. Then he disappeared until Dec. 17, 1936, when he came back so weak that he could walk only with difficulty. He had the dermatitis, stomatitis, diarrhea and

peripheral neuritis which accompany pellagra. He had continued his intake of alcohol, but instead of taking 4 tablespoonfuls of powdered brewers' yeast daily he had been using only a few tablets of powdered brewers' yeast daily. Vision remained 20/20 in each eye, and there was only the same small residual scotoma between the blindspot and the fixation area in each eye (fig. 3 *B*). He was again hospitalized and given powdered brewers' yeast in doses of 2 tablespoonfuls

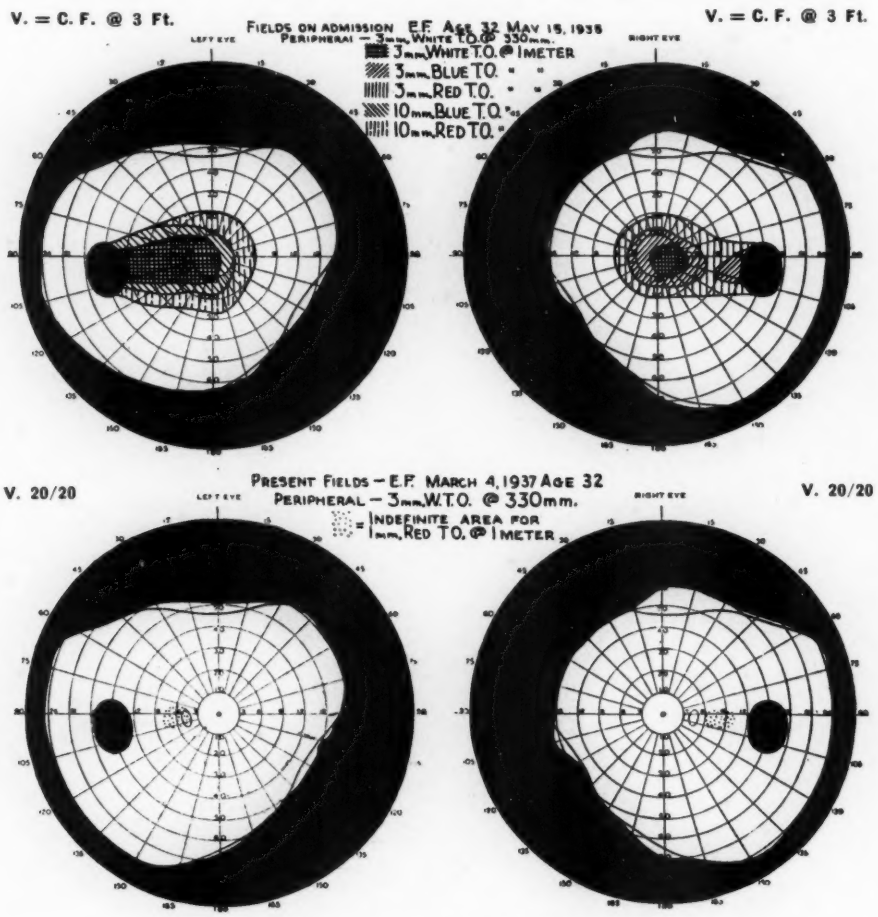
V. = C. F. @ 3 Ft.
FIELDS ON ADMISSION E.F. AGE 32 MAY 15, 1936
PERIPHERAL — 3mm. WHITE T.O. @ 330mm.
3mm. WHITE T.O. @ 1METER
3mm. BLUE T.O.
3mm. RED T.O.
10mm. BLUE T.O.
10mm. RED T.O.
V. = C. F. @ 3 Ft.
V. 20/20
PRESENT FIELDS — E.F. MARCH 4, 1937 AGE 32
PERIPHERAL — 3mm. W.T.O. @ 330mm.
= INDEFINITE AREA FOR 1mm. RED T.O. @ 1METER
V. 20/20

Fig. 3 (case 5).—Fields of E. F.

five times daily (90 Gm.), wheat germ in doses of 4 tablespoonfuls three times daily, vegex in doses of 1 teaspoonful three times daily and 5 or 10 cc. of liver extract [9b] intramuscularly daily. He was also given 1 pint of liquor daily. Gastric analysis showed lack of free hydrochloric acid. His condition again rapidly improved, and he was discharged in four weeks free from any symptoms. Since then he has continued his usual consumption of tobacco and alcohol but has faithfully taken 2 tablespoonfuls of yeast daily. He has no symptoms.

CASE 6.—H. R., a 63 year old man, was first seen in the Vanderbilt Clinic on May 29, 1935. His vision at that time was 20/200 in each eye with correction. The right eye had not had good vision since having a corneal ulcer twelve years previously. It showed diffuse corneal opacities and an incipient cataract. The left eye also had an incipient cataract, which was not sufficiently dense to explain the decreased vision The disks appeared normal, and the fields showed typical

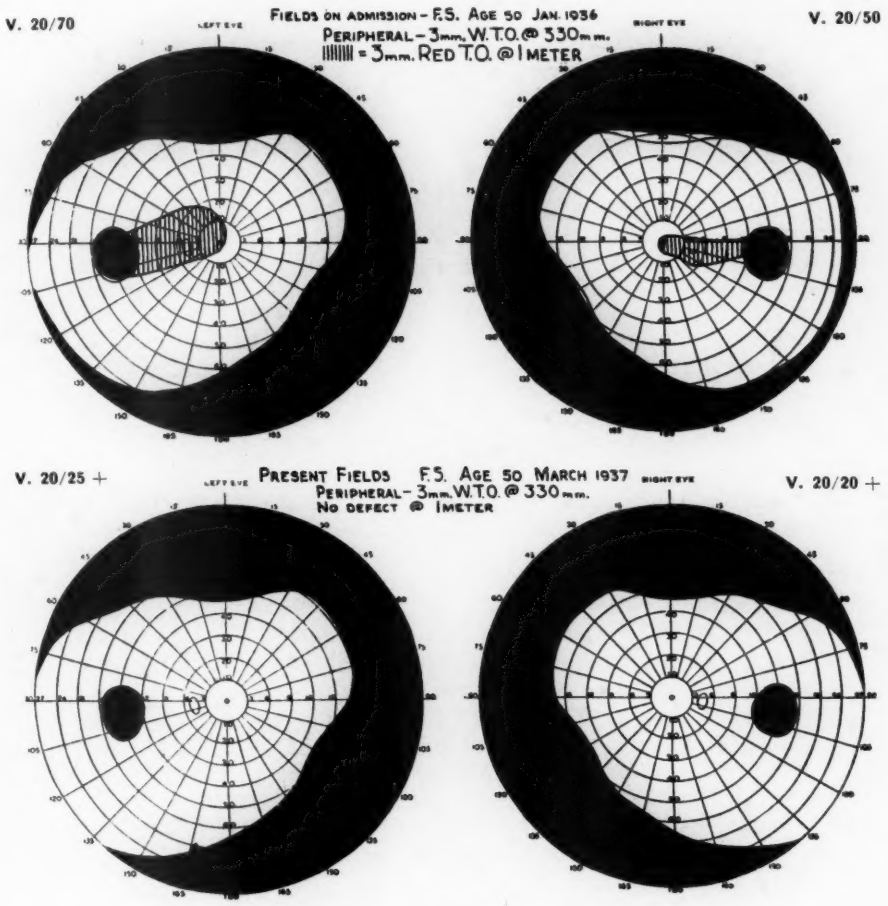
V. 20/70
FIELDS ON ADMISSION - F.S. AGE 50 JAN. 1936
PERIPHERAL - 3mm. W.T.O. @ 330mm.
= 3mm. RED T.O. @ 1 METER
V. 20/50
V. 20/25 +
PRESENT FIELDS F.S. AGE 50 MARCH 1937
PERIPHERAL - 3mm. W.T.O. @ 330mm.
NO DEFECT @ 1METER
V. 20/20 +

Fig. 4 (case 7).—Fields of F. S.

centrocecal scotomas. The patient drank four glasses of beer and smoked from ten to twelve cigars daily. He was advised to discontinue tobacco and alcohol and did so, but when I first saw him two months later, on July 26, vision had decreased to 10/200 in the right eye and to 5/200 in the left. He was admitted to the Institute of Ophthalmology and allowed to drink and smoke as much as he wished—this was about ten cigars daily and from 6 to 14 ounces (177 to

414 cc.) of liquor. He took all the prescribed diet. No improvement was seen for almost a month; then it came gradually. In two months vision in the left eye had improved from 5/200 to 20/80. The opacities in the cornea and lens of the right eye appeared sufficient to explain the poor vision of 10/200 in that eye. In three months vision of the left eye was 20/60—, and on discharge from the hospital vision of the right eye was 10/200 and that of the left eye 20/40. There was marked decrease in the density of the scotomas. This patient remained in the hospital under close observation for a total of five months. During this time vision of the left eye, which was the only eye in which vision could be expected to improve, increased from 5/200 to 20/40. Except for one week, during this period he smoked an average of ten cigars daily and drank from 6 to 14 ounces of liquor daily. On discharge from the hospital he was advised to decrease or discontinue the use of tobacco and alcohol. It is not certain whether he followed this advice. Fourteen months since discharge vision of the left eye is the same—20/40+.

CASE 7.—F. S., a 50 year old unemployed Italian bank clerk, was referred to me for treatment by Dr. Charles Perera, who made the diagnosis of toxic amblyopia. Vision corrected was 20/50 in the right eye and 20/70— in the left; typical centrocecal scotomas were present (fig. 4 *B*). The disks were normal in appearance. The patient stated that his vision had been gradually decreasing for the past year. He was accustomed to drinking between 1 pint and 1 quart of wine and smoking one package of cigarets daily. For the past several years he had noticed some abdominal discomfort and during the past year had lost from 10 to 15 pounds (4.5 to 6.8 Kg.) in weight. His appetite was poor. On Jan. 20, 1936, he entered the Institute of Ophthalmology. He brought his own wine and cigarets with him, and throughout his stay in the hospital he used the same amount of wine and smoked the same number of cigarets that he had consumed while the tobacco-alcohol amblyopia was developing. He remained in the hospital under close observation for seven weeks. He took the prescribed diet. His weight increased 15 pounds (6.8 Kg.). His vision gradually improved to 20/20— in the right eye and to 20/30— in the left in seventeen days and was 20/20+ in the right eye and 20/30+ in the left when he left the hospital. Since then fourteen months have passed. At no time during this long course of observation has the consumption of alcohol or tobacco ever been reduced. He has continued to take 1 or two tablespoonfuls of yeast daily because I was not sure whether or not it would be safe for him to discontinue it. Vision now is 20/20+ in the right eye and 20/25+ in the left. I am unable to plot any scotoma in either eye (fig. 4 *B*).

ABSTRACT OF DISCUSSION

DR. ARTHUR M. YUDKIN, New Haven, Conn.: It is noteworthy that two distinct types of therapeutic measures should be offered at this meeting for the cure of the same ocular disturbance. Tobacco-alcohol amblyopia has been cured by other methods. One, in particular, is to eliminate the consumption of alcohol and tobacco and give the patient a well balanced diet. This method does not restore vision until the patient has adhered to the regulations for many months. At the Milwaukee session of the American Medical Association in 1933 I recommended the treatment of this disease on the basis of a well balanced diet supplemented by cod liver oil, and in some instances vitamin B complex was also given. Most of the patients improved, even though they consumed a moderate amount of alcohol and tobacco. When destruction of the

optic nerve was present, it was difficult to restore vision. Frequently a constitutional disturbance, such as diabetes, delayed the healing process. Dr. Carroll has presented a similar observation. In spite of his ability to cure this condition while the patient consumes his regular amount of alcohol and tobacco, Dr. Carroll realizes the advantages of diminishing and even eliminating the drugs.

From a scientific standpoint this paper is a valuable contribution, for it demonstrates that alcohol and tobacco when consumed by a person having a lowered resistance may produce not only a general condition but also ocular defects. The severity of the clinical picture depends on the general health of the patient and the amount of alcohol or tobacco consumed. Many cases are recorded of the appearance of vitamin B complex deficiency associated with voluntary restriction of food as a result of digestive disturbance, or as a therapeutic measure, as in diabetes, in pregnancy attended by vomiting, in alcoholism and in other conditions. Strange as it may seem, many deficiency diseases are now cured by the addition of large doses of vitamin B complex to the diet. Nevertheless it cannot be said that alcohol-tobacco amblyopia is due to vitamin B complex deficiency.

From experimental work, it can readily be seen why one type of symptoms appears in one group of animals and another type in still another. Dr. Cowgill and his collaborators kept one group of dogs on a minimum amount of vitamin B_2 (G), while another group was deprived of B_2 (G) entirely. The first group showed collapse, whereas the latter group suffered eventually from ataxia. Two quite different syndromes were observed. The picture of complete deprivation was different from that of what might be called subnutrition. That is probably why in one group beriberi appears, in another pellagra, and in still another ocular disturbance. It becomes evident that a person suffering from a deficiency may at some time or other have different nerves involved. In dealing with man it must not be forgotten that the intestinal tract plays a great role in the distribution of materials necessary for the upkeep of the body. The strains of modern life have produced certain nervous manifestations that have actually upset the whole gastro-intestinal mechanism, with the result that the food is not properly prepared for digestion and absorption. I recommend that the ophthalmologist not only treat his patients with local medication but become interested in the general health of his patient. If he is confident of his medical judgment, there is no reason why he should not advise the patient concerning diet and the mode of living.

In spite of the dramatic results obtained in tobacco-alcohol amblyopia by Dr. Carroll, I cannot subscribe to the continued use of these drugs except in exceptional cases, nor can I see the reason for such large amounts of vitamin B complex except for investigative purposes. Excellent results may be obtained in this ocular disturbance, which, I believe, is associated with a dietary deficiency probably of a gastro-intestinal nature, by the restriction of alcohol and tobacco, by the elimination of constitutional complications and by prescribing a sensible, well balanced diet supplemented with moderate doses of standardized brewers' yeast and properly assayed cod liver oil.

Dr. Frank Carroll, New York: The idea of Dr. Duggan that tobacco amblyopia is caused by a spasm of vessels supplying the optic pathway deserves critical consideration. There is some evidence that smoking constricts the peripheral vessels. A report published recently, however, indicates that there is no more peripheral vasoconstriction from smoking than there is from taking a deep inspiration of pure air. Several of my patients with toxic amblyopia who were heavy smokers have been tested by members of the department of pharmacology of Columbia University, and no evidence was found by an accurate plethysmograph method of any peripheral vasoconstriction. But even if smoking does tend to constrict the peripheral vessels and raise the blood pressure, this does not indicate that either the cerebral or the retinal vessels are constricted. In fact, just the reverse is apt to be true. For example, when the epinephrine is given, vasoconstriction of the peripheral vessels occurs. This causes an increased blood pressure with resulting vasodilatation of the cerebral vessels. Lambert has shown that in animals epinephrine, which causes constriction of peripheral blood vessels, tends to cause vasodilatation of the retinal vessels because of the increase in the blood pressure. If there should be a spasm, where would it be and what would be its nature? It is almost certainly not in the retina; the retinal vessels appear perfectly normal in many of these patients. The pathologic change is essentially limited to the papillomacular bundles, and therefore this hypothetic spasm would have to be limited to the vessels supplying the papillomacular bundle. The chief vessel supplying the papillomacular bundle is the central retinal artery, and when there is an ordinary spasm of this vessel it can be seen with an ophthalmoscope. It does not resemble anything seen in tobacco amblyopia. Moreover, this type of amblyopia comes on gradually and disappears gradually. Spasms of the central retinal artery or its branches that are familiar come on suddenly; therefore, one is expected to assume a special kind of spasm foreign to the present knowledge.

Dr. Duggan has stated that in tobacco-alcohol amblyopia moderate drinkers improve more rapidly than nondrinkers, and he thinks this is interesting in view of the vasodilating property of alcohol. How would he explain the existence of a disease, indistinguishable from tobacco-alcohol amblyopia in patients who smoke little or not at all, but who are heavy drinkers? Certainly alcohol is not a vasoconstrictor. According to this vascular theory, my patients with this disease who were heavy drinkers would have been especially immune because they were heavy drinkers and should never have had amblyopia—but they did. Vasodilators are suggested as being beneficial. One would expect a vasoconstrictor such as epinephrine to be of more value. This would raise the blood pressure and thus dilate the cerebral and retinal vessels as well as increase the rate of blood flow through the brain. Dr. Duggan has stated that the speed of recovery of patients receiving vasodilators is more rapid than that of patients not receiving them. This is a difficult point to prove and needs controls. An attempt has been made to use other cases reported in the literature as controls, but they are not comparable. If Dr. Duggan is to persist in his efforts to

prove the value of these drugs, he should treat every alternate patient with an equal number of injections of saline solution or even vasoconstrictors.

I have the records of nine patients with this condition who were treated with intravenous injections of sodium nitrite either by other members of the hospital staff or by myself during the past five years. An effort was made to give the vasodilator exactly as suggested by Dr. Duggan. Some patients received daily injections; others received injections twice daily. The average number of injections received by each patient was fifty. Two of the nine patients showed no improvement. The others gradually became better. I am sorry to say that there was nothing to indicate that the vasodilator had hastened the rate of recovery.

Dr. Yudkin suggested that perhaps I was using larger doses of brewers' yeast than necessary, and that may possibly be true. I do believe that large doses are essential if the patient continues to drink and smoke. One of my patients stopped taking what I suggested and took yeast cakes instead, and pellagra developed within a month. He then took the powdered brewers' yeast again, and the pellagra cleared up. He then failed to follow my advice and took tablets of brewers' yeast instead of taking powdered yeast in tablespoonful doses, and the pellagra returned. It rapidly cleared up again on the suggested dosage.

DR. WALTER F. DUGGAN, New York: I have read most of the reports that Dr. Carroll has read concerning the vasoconstrictive effect of smoking on the peripheral vessels and am amazed at the conflicting statements. I have treated with vasodilators alone some fifty patients with tobacco amblyopia without atrophy of the optic nerve. The visual improvement has been so rapid in many cases that I have been surprised. Dr. Fralich and Dr. Bracken, of New York, have obtained good results. I have only the improvement in vision, the patient's word and the decrease in the size of the scotoma on which to base my figures. As to where the spasm is located, Dr. Carroll mentioned that the papillomacular bundle is involved and that it is supplied by the central artery of the retina. It is, in the retina, but the central artery of the retina usually does not supply any fibers of the optic nerve posterior to the point at which it enters the optic nerve, and it certainly does not supply the papillomacular bundle in the chiasm. Behr recently mentioned that the central artery of the retina is strictly a retinal artery as soon as it enters the optic nerve. My feeling is that the arterioles which are involved are in the region of the chiasm, because of the bilateral field defects. I have seen no change, as a rule, in the retinal vessels after this treatment, but I have seen an improvement in vision and a decrease in the size of the scotoma. Tobacco amblyopia does not come on suddenly. Amblyopia does come on suddenly in cases of retrobulbar neuritis, and I feel that this is much more analogous to a spasm of the central retinal artery than toxic amblyopia is. As to why these particular vessels (in the chiasm?) are involved, I have no explanation other than that they are end-arterioles. I think that Dr. Carroll's cases

and my cases belong to different groups. In the nine cases that he reported in December 1936 the average age was 36. The average age which has been found by all authors in cases of tobacco amblyopia is between 50 and 56 years. In cases of real tobacco amblyopia the patient does not show polyneuritis or symptoms of pellagra. In the cases which Dr. Carroll reported the patients did show symptoms of a deficiency disease. I think that he and I are discussing different disease entities.

INTRA-OCULAR INVASION BY THE LARVA OF THE ASCARIS

REPORT OF A CASE WITH UNUSUAL COMPLICATIONS

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It is a rare occurrence for the interior of the eye to be invaded by any parasite. Of those parasites classified as the nematodes, the filaria has been found within the eye by Wright and his collaborators,[1] Fernando[2] and others, and Fulleborn[3] stated the belief that the retinal hemorrhages seen in hookworm infections are the results of embolic embryonic larvae of the uncinaria.

The ascaris (Ascaris lumbricoides) is one of the most common of the nematoda, and Ward[4] commented in his monograph on ocular parasites that it is only indirectly parasitic to the eye, such as through some constitutional ailment. However, this intestinal round-worm (Ascaris) has been found in its young or adult form in many organs of the human body, even in the brain and in the heart,[5] and it seems reasonable to believe that through the same channels, the circulation or the lymphatics, it might gain access to the interior of the eye.

Read at the Seventy-Third Annual Meeting of the American Ophthalmological Society, Hot Springs, Va., June 4, 1937.

1. Wright, R. E.: Adult Filaria (Wuchereria Bancrofti) in Anterior Chamber, Brit. J. Ophth. **18**:646, 1934. Wright, R. E.; Iyer, P. V. S., and Pandit, C. G.: Description of an Adult Filaria (Male) Removed from the Anterior Chamber of the Eye of Man, Indian J. M. Research **23**:199, 1935.

2. Fernando, S. E.: Ocular Filariasis (Adult *Wuchereria bancrofti*) in the Anterior Chamber of Human Eye, J. Trop. Med. **38**:17, 1935.

3. Fulleborn, F.: Untersuchungen über den Infektionsweg bei Strongyloides und Ankylostomum und die Biologie dieser Parasiten, Arch. f. Schiffs- u. Tropen-Hyg. (supp.) **18**:182, 1914.

4. Ward, H. B.: Ocular Parasites, in Wood, Casey A.: American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1918, vol. 12, p. 9299.

5. Boettiger, C., and Werne, J.: Ascaris Lumbricoides Found in the Cavity of the Human Heart, J. A. M. A. **93**:32 (July 6) 1929. Sterling, R., and Guay, A. J. L.: Invasion of the Female Generative Tract by Ascaris Lumbricoides, ibid. **107**:2046 (Dec. 19) 1936.

The experimental work of Suyemori [6] supports this belief, for after feeding guinea-pigs the eggs containing fully developed embryos, "ascaris larva in the ocular organs were seen moving with blood—30 hours after infection," and were later found in the retina and optic nerve and in abundance in the uveal tract.

The case to be reported is one of intra-ocular invasion by a larva. Its position on the lens offered an excellent opportunity for study with the slit lamp and the microscope, and it was classified as the larva of an ascaris. I am conscious that for scientific accuracy this opinion might be challenged, as the larva could not be removed for dissection and microscopic study, yet the evidence is so strongly in favor of it being the larva of an ascaris that this unique case is reported.

REPORT OF CASE

History.—On Dec. 1, 1936, J. Y., aged 8, was referred by a practitioner in a rural community of Northern Georgia, the opinion being offered that the patient had acute glaucoma. The eye had been inflamed and painful for three days; there was no history of trauma, and the father reported that so far as he knew the boy was healthy. The patient had never been outside of his county. The father, a fairly intelligent farmer, gave a good account of himself and his large family. The only information which was of value (then it was thought to be inconsequential) was that two other children had been recently treated for "worms."

Ocular Examination.—The right eye had vision permitting perception of movements of the hand. The globe was intensely injected. The cornea was steamy, with several keratitis dots on the posterior surface; the anterior chamber was deep, and the aqueous was cloudy. The iris was off color; the pupil was irregular, semidilated and blocked by a thin exudate which prevented an ophthalmoscopic examination. The intra-ocular tension was 37 mm. of mercury (Schiötz). The external and the internal examination of the left eye gave normal results.

An opinion was expressed that the patient had acute iridocyclitis associated with secondary glaucoma of undetermined origin, and as the father would not consent to his son remaining, drops of atropine sulfate and hot packs were ordered, with the caution that the home physician should closely watch the eye.

One week later the eye was moderately injected; the pupil was widely dilated except for dense adhesions at 9 o'clock, and only a fundus reflex was obtained. The tension by palpation was normal.

In two weeks the eye was comfortable and rapidly clearing. The pupillary area was now clear except for the adhesions at 9 o'clock and some scattered pigment on the anterior surface of the lens. The lens was found to be dislocated temporarily, and in the adjacent circumlental space there were exudates which extended into the vitreous. The tension was plus one.

I suspected that the eye had been traumatized, but the patient and his father were equally positive that such was not the case.

In the fourth week the patient was still comfortable, and the globe was only slightly injected. The tension was plus one. The cornea was faintly edematous;

6. Suyemori, S.: On the Pathological Changes in the Eye During the Incipient State of Infection with Ascaris (Experimental Study), Taiwan Igakkai Zasshi, 1925, vol. 1, no. 239.

the iris was dilated and tremulous, and the exudates in the circumlental space had increased. Coming from behind the iris at 5 o'clock and lying on the anterior surface of the lens was seen for the first time a slender, translucent pearly white object, which had the appearance of a thin delicate spear point, for the sides were beveled and a distinct central line coursed down from its rounded end to where

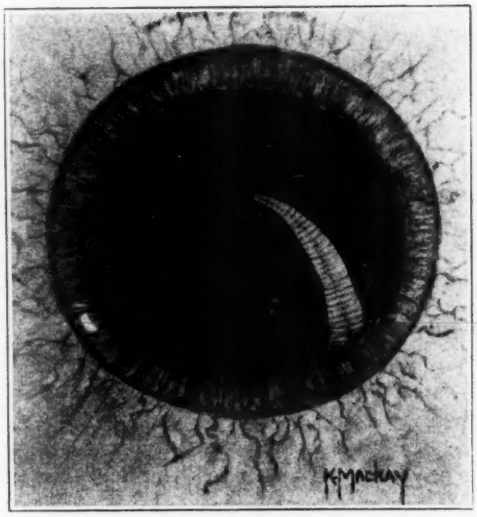

Fig. 1.—Larva of Ascaris on the anterior surface of the lens.

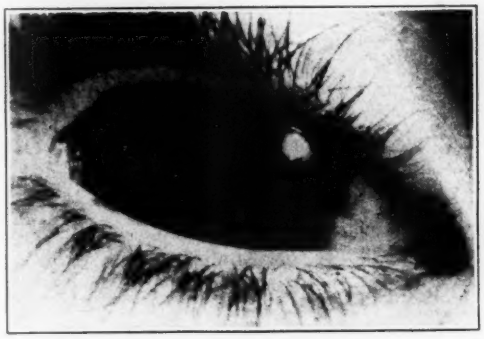

Fig. 2.—Unretouched photograph of the larva of Ascaris.

the object passed behind the iris. The object was nonmotile, had a slight slant temporalward and measured 3 mm. long, and at the pupillary margin its maximum width was 1 mm. It was suggested that this object was the larva of an intestinal parasite. The following day several stools were examined, but no ova were found.

In the fifth week the eye was very quiet, and the tension was normal. The larva had decidedly grown and measured 1.5 mm. by 5 mm. It had assumed a distinctly curved position and was now definitely round. Throughout its entire

length were delicate horizontal lines, and running down its center was a brownish marking. When the globe was rotated downward one could readily see with the ophthalmoscope that the larva curved around the equator of the dislocated lens and extended 2 mm. beyond; it was attached to the posterior surface by its head. The fundus could now be seen for the first time, and it was found to be normal. The opacities in the vitreous were entirely confined to the lower nasal sector.

Dr. Thomas F. Sellers, parasitologist of the Georgia State Board of Health, was asked to see the eye, and he expressed the opinion, after making several slit lamp examinations, that the larva had all the morphologic characteristics of an immature ascaris.

The patient was hospitalized for a complete study; the results of all the examinations were normal. Repeated examinations of the stools showed no ova of intestinal parasites. Examination of the blood showed 75 per cent hemoglobin, 4,340,000 red cells, 8,200 white cells, 51 polymorphonuclears, 47 lymphocytes and 2 eosinophils. Smears gave negative results.

With the knowledge that the cuticle and celomic fluid of the ascaris is often toxic and as the eye was becoming more quiet, it was decided that the best policy would be to wait for developments, in the hope that the larva might find its way into the anterior chamber, from which it could be more readily removed. Treatment with drops of atropine sulfate and hot packs was continued.

At the patient's visit in the sixth week the eye was still quiet, and the tension was normal. The body of the larva appeared to be less glistening, and scattered over its surface and on the adjoining capsule of the lens were seen delicate brownish markings, which, when viewed with the slit lamp, resembled the curled-up bark of a silver birch tree; these were interpreted as being cast-off particles of desquamated cuticle. The size of the larva had not changed, and it was believed that it was disintegrating.

The eye remained quiet until the eleventh week, when the patient returned with the statement that it had been inflamed for the past four days but there had been no pain. The globe was quite congested, the aqueous cloudy, the iris off color, and the pupil slightly contracted, in spite of the daily use of atropine sulfate. The iris was adherent to the larva on the nasal side, and the detritus coming from the desquamated cuticle had increased.

The tension of the right eye was 10 mm. of mercury, and that of the left eye was 17 mm.

It was now thought that the acute flare-up of iritis was due to the disintegration of the larva and that the best procedure would be to remove it.

Operation.—With the patient under induced anesthesia with ether, a limbal incision was made with a thin knife from 4 to 7 o'clock. This approach was decided on for the reason that if the texture of the larva was reasonably tough, as is that of the adult worm, it could be grasped and teased out without further dislocation of the lens or loss of vitreous.

An attempt was made to grasp the larva with a capsule forceps, but, to my astonishment, its substance was like jelly; only fragments could be removed, and there remained the tail, delicate fragments on the surface of the lens, the part behind the iris and the head. The anterior chamber was gently irrigated with saline solution, in the hope that these particles might flow out, but they were firmly adherent to the lens. As the iris tended to prolapse, a drop of solution of

physostigmine was used and a firm bandage applied. At the first dressing the globe was found to be quiet, but the iris had prolapsed. This was excised, and the pillars were replaced.

Outcome.—The eye made a prompt recovery, and when the patient was last seen, on June 27, seven months after the onset, vision was 10/200, and the tension was normal. The pupil was dilated, and there was a small keyhole-shaped coloboma of the iris at 5 o'clock. The old adhesion of the iris at 9 o'clock was still present, and on the anterior capsule were a few deposits of pigment. No trace of the larva could be seen on the anterior or the posterior surface of the lens. Several fine lines were seen in the cortex of the dislocated lens, and a few vitreous opacities remained.

COMMENT

In the case which is presented every symptom is referred to the affected eye, and the evidence is that the iridocyclitis and even the dislocation of the lens were the direct results of intra-ocular invasion by a parasite, which was classified as the larva of an ascaris.

The mode of infection and the cycle of development of the ascaris may be recalled with some interest. The ova are carried by contaminated food into the intestinal tract, where the larva are liberated by the hatching of the eggs in the small intestine and then penetrate the portal circulation. After a brief stay in the liver they pass through the right side of the heart into the lung and undergo further development in the capillaries; thence they pass into the vesicles, up the trachea, and down the esophagus into the stomach and intestine, where they develop into adult worms.

At the outset one must assume that the patient had a mild parasitic infection, notwithstanding the fact that no ova were found in the stools. The fact that stools contain no ova does not always exclude parasitic infection. It is known that the patient was exposed to the same home influences which caused two other children in the family to be treated for "worms."

The fact that no other case of intra-ocular invasion by the larva of an ascaris has been reported should not deter one in the belief that it is impossible. On the contrary, such an invasion would seem most logical, since the ascaris has been found in other organs of the body, notably, the heart and brain. It is difficult to say whether this living embolus passes through the lymphatics or the arterial circulation, although it is possible to conceive that an embryonic larva while in the lungs during the period of migration could pass through the pulmonary capillaries into the general circulation and, in cases in which the eye is involved, gain entrance through any penetrating vessel.

Since the iris and ciliary body were early involved in this case, one may assume that an anterior ciliary artery, located approximately

at 5 o'clock and supplying those structures, was the carrier. However, the larva might have entered at 9 o'clock, the site of the dense adhesion of the iris, left its infection and migrated. It finally attached itself by the head to the posterior surface of the lens and extended its caudal end into the proximal fibers of the suspensory ligament, which were likely torn or worn away for a considerable extent by its motility and growth. The destruction of the nasal zonular fibers, combined with the traction of the dense adhesions in the temporal portion of the iris, offers a reasonable explanation for the temporal displacement of the lens noted at the third visit, one week before the larva made its appearance. Indeed, one may speculate further and suggest that this dislocation may have been one reason why the infection was largely limited to the iris, as it allowed the larva to escape forward through the large circumlental space instead of being confined to the ciliary region.

It is commonly known among parasitologists that the cuticle and the celomic fluid of the ascaris (especially Ascaris megalocephalia) are often toxic, and it was thought that any attempt to remove the larva during the period of the acute ocular infection would have added injury to the globe. For that reason and because the inflammatory symptoms were rapidly subsiding, it was deemed best to postpone any operative procedure, in the hope that the larva might free itself and fall into the anterior chamber.

In the fifth week it was noted that the larva was dying and undergoing degeneration, as evidenced by lack of motility, no increase in size and especially desquamation of the cuticle. A week later there was a recurrence of the iritis, and it was then thought that the globe could best be preserved by removal of the larva. What happened at the operation is recalled in the report of the case.

The fact that the eye showed no reaction after the operation, even when fragments of the larva remained, now leads me to believe that the wisest procedure would have been not to disturb it and that in time the larva would have been completely absorbed. There is no precedent for this opinion, except in Anderson's [7] report of a case of ophthalmomyiasis interna, in which the larva of an insect having migrated into the vitreous, absorbed after eighteen months, with no injury to the eye.

In attempting a differentiation of the types of larva which would present pictures resembling the one in this case, two possibilities can be considered; the larva of the fly and the larva of a nematode.

The former type should be ruled out, as this larva is always segmented and its surface in no way resembles the round, smooth surface

7. Anderson, W. Banks: Ophthalmomyiasis, Am. J. Ophth. **18**:699, 1935.

of the worm that has been described. Besides there is often the history of the insect striking the eye and becoming lodged in the fornix, with the usual discomfort, which was not present in this case.

In regard to the latter type, I present the report of Dr. Thomas F. Sellers, parasitologist of the Georgia State Board of Health, who aided in the preparation of this paper.

"The morphologic features of the foreign body, as described by Dr. Calhoun, were characteristic of the caudal end of a larva of a nematode. The tapering contour ending in a rather sharp point, the smooth cuticle showing only faint transverse striations, the slender lightly pigmented central canal—all these characters were present.

"The common nematodes parasitizing man are: the hookworm (Uncinaria americana and Ancylostoma duodenale), the pinworm (Oxyuris vermicularis), the whipworm (Trichuris trichiura), the round-worm, or the stomach worm (Ascaris lumbricoides), the trichina (Trichinella spiralis), Strongyloides stercoralis and several species of filaria. Of these, the filaridae, especially Filaria loa, have been known to parasitize the eye. That one was not dealing with the filaria in this case is evident, for none of the morphologic characters distinctive of the microfilaria were present, and in man this parasite is exceedingly rare in the interior of this country. The larva of the pinworm and that of the whipworm complete their life cycles from egg to adult entirely within the intestinal tract. They never invade the blood stream. The larva of the trichina does invade the blood stream and has been found encysted in the ocular musculature. This larva, however, is entirely too small to be considered in this case. The larva of Strongyloides likewise invades the blood stream, but it, too, is almost microscopic in size.

"This leaves for consideration only the hookworm and round-worm. The life cycles of these are somewhat similar. It is probable that some of these larvae, which are very minute, fail to escape to the air passages, and continue on into the pulmonary arterioles and thence back to the heart, to be distributed via the arterial circulation to any part of the body. Just how far such a larva thus entrapped could develop is not known. Nevertheless, one did develop in this case to such size as to protrude a good half its length around the lens and into the anterior chamber, so that 5 mm. of its length was plainly visible. The total length of the worm must have been at least 10 mm., perhaps 15 mm. Since this approximates the length of the young adult hookworm and yet this parasite had none of the characters of an adult form, it is believed that the hookworm can also be eliminated. Therefore, my opinion is that one was dealing with an entrapped larva of an ascarid, which, after progressing to a certain point in its unnatural environment, died and was in process of decomposition when Dr. Calhoun operated."

SUMMARY

The case of a young boy is presented, with an account of the following train of symptoms, recorded at weekly intervals, and my conclusions.

In the first week there were acute iridocyclitis and secondary glaucoma.

In the second week the eye was more quiet, and the tension was normal.

In the third week the lens was dislocated.

In the fourth week the appearance of a larva on the anterior surface of the lens was noted.

In the fifth week the larva had almost doubled its size.

In the sixth week there was evidence that the larva was dying and disintegrating.

In the eleventh week there was an acute flare-up of the iritis. The larva was definitely disintegrating, and an attempt to remove it was unsuccessful.

In the twelfth week the eye was quiet.

In the seventh month the eye was quiet. Complete absorption of the larva had occurred. The larva was identified (by T. F. S.) as an immature ascaris.

This experience now leads me to believe that the larva would have absorbed had it not been disturbed.

NEUROGENIC ORIGIN OF CHOROIDAL SARCOMA

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The main object of this paper is to show that the neoplasm of the choroid which has hitherto been described in the literature as choroidal sarcoma is much more probably a neurogenic tumor, arising from the Schwann sheath cells of nerves traversing the choroid. The neural origin of so-called sarcoma of the choroid will be supported by the histologic observations in seven cases which I have studied. The work, so far as I know, is new.

Before presenting and discussing my histologic studies, it is desirable to summarize briefly what the literature shows concerning neurogenic tumors in general, as well as to glance at the neurologic structure of the choroid and the neural and other neoplasms that are found in this vascular membrane.

NEUROGENIC NEOPLASMS IN GENERAL

Neoplasms showing more or less nerve tissue in their structure have been known for a long time. Tumors, especially those of the nervous system, composed of pure ganglion nerve cells and nerve fibers occurring in the course of a nerve have been called neuroma. Their existence has been denied by some, but Bruce and Dawson [1] reported them in scattered nerve tissue as embryonic rests.

More often tumors, single or multiple, containing nerve tissue have been found occurring along the course of nonmedullated, deep-lying nerves associated with fibrous tissue. Some show a preponderance of neural tissue and approach the neuroma type. Others are richly cellular, showing all kinds of spindle cells arranged in intertwining fascicles.

The cellular unit of the nervous system is the neuron, composed of a cell body, dendrites and a neurite or axon, which ends in teledendrons. Most of the nerves contain only neurites and no cell bodies, but some peripheral ganglionated plexuses of the sympathetic nervous system have nerve cells scattered among the nerve fibers. Peripheral nerves are medullated; deep-lying nerves are not. The neurite in medullated

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Read at the Seventy-Third Annual Meeting of the American Ophthalmological Society, Hot Springs, Va., June 4, 1937.

1. Bruce, A., and Dawson, J. W.: Multiple Neuromata of the Central Nervous System, Rev. Neurol. & Psychiat. **11**:235, 1913.

nerves is covered by myelin, a product of the nerve cell; outside of the myelin, which is absent in nonmedullated nerves, is the protoplasmic envelop, the sheath of Schwann, consisting of a syncytium of cells which migrate from the neural crest in early embryonic life. Some doubt has existed as to the nature of the sheath of Schwann. Harrison,[2] I believe, was the first to show its ectodermal origin. Penfield [3] described it mesodermal connective tissue. Recent studies, however, seem to have confirmed its ectodermal nature. The three structures of the peripheral nerve proper—the neurite, the myelin and the sheath of Schwann—are therefore taken here as ectodermal in origin. The whole nerve fiber is embedded in connective tissue, the endoneurium and perineurium, which are admittedly mesodermal. The pivotal importance of the ectodermal origin of the Schwann sheath cells to the histogenesis of neurogenic neoplasms will presently appear.

The uncertainty of pathologists regarding the nature of neurofibrous tumors is expressed in the variety of their names. They are known as Recklinghausen's fibromatosis, neurinoma, sympathetic blastoma, fibroma and neurofibrosarcoma and by other names. The tumors may arise along the course of either medullated or nonmedullated nerves.

Ewing [4] said that in actively growing neurosarcoma the cells are very numerous, are small, spindle shaped or round and are devoid of definite stroma. Kaufmann [5] stated that sarcoma composed largely of spindle cells may develop as the cell-rich malignant variety of neurofibroma from the connective tissue of nerves. Mallory [6] and other American pathologists agree that neural tumors may arise from the supporting endoneural connective tissue and therefore are mesodermal in origin. In the recent edition of his "Text-Book of Pathology" MacCallum,[7] admitting the ectodermal origin of the Schwann sheath cells, expressed the opinion that in proliferation these cells in neurinoma revert to an embryonic stage in their development, since they proceed to excessive growth which only passively includes nerve fibers.

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2. Harrison, R.: Neuroblast Versus Sheath Cells in the Development of Peripheral Nerves, J. Comp. Neurol. **37**:123, 1924.

3. Penfield, W.: (*a*) Encapsulated Tumors of the Nervous System, Surg., Gynec. & Obst. **45**:178, 1927; (*b*) Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932.

4. Ewing, J.: Neoplasic Diseases, ed. 3, Philadelphia, W. B. Saunders Company, 1928, pp. 186, 466, 167 and 931.

5. Kaufmann, E.: Lehrbuch der speziellen pathologischen Anatomie, ed. 8, Berlin, W. de Gruyter & Co., 1922, p. 1566.

6. Mallory, F. B.: Type-Cell of the So-Called Dural Endothelioma, J. M. Research **41**:349, 1920.

7. MacCallum, W. G.: A Text-Book of Pathology, ed. 6, Philadelphia, W. B. Saunders Company, 1936, p. 1045.

The view that the neurofibrous tumors, especially neurosarcoma, arise from the supporting endoneural connective tissue, was generally held until 1910, when Verocay [8] published a report in which he showed clearly that they are derived from proliferation of Schwann sheath cells. Masson,[9] of Montreal, has published two important and convincing histologic studies in which he confirmed and extended Verocay's work, clearing up much misconception of neural neoplasms. In the first paper,[9a] published in 1926, he proved conclusively that pigmented cutaneous nevi are in reality neural tumors with preponderance of proliferation of Schwann sheath cells. Furthermore, he noted the Wagner-Meissner type of tactile corpuscles in schwannian tumors and concluded that the plexus of Schwann sheath cells which composes the greater part of nevi proceeds from sensory nerves. He contended further that in certain nevi the Schwann cells can fabricate melanin; judging by the behavior of melanoblasts in nevi, he attributed a neural nature to these pigmented cells, thus affording a point of view differing from that of the epithelial or connective tissue origin of melanoblasts in the skin.

I must pause here to remark somewhat on melanotic neoplasms generally. Melanotic sarcoma or melanoma was first described in 1784 as occurring in horses. Laënnec was the first to write on this type of tumor in man. Ever since, according to Coley and Hoguet,[10] discussion has been continuous as to the origin of melanotic tumors, the question being whether they arise from connective tissue or from epithelial tissue. Outside of the uveal tract they usually develop from congenital moles or pigmented warts. The majority of investigators at present agree with Masson and his followers that they are ectodermal in origin; however, Coley and Hoguet expressed the view that some melanotic tumors originate from connective tissue.

Nitsch,[11] of the Vienna Eye Cinic, said that pigmented tumors arise by preference from the sympathetic nerves. Parsons [12] observed that in the case of melanotic tumors there can be no doubt that the pigmented cells arise from the chromatophores, and he considered pigmented

8. Verocay, J.: Zur Kenntnis der Neurofibroma, Beitr. z. path. Anat. u. z. allg. Path. **48**:1, 1910.

9. Masson, P.: (*a*) Les naevi pigmentaires, tumeurs nerveuses, Ann. d'anat. path. **3**:417 and 657, 1926; (*b*) Experimental and Spontaneous Schwannomas, Am. J. Path. **8**:367, 1932.

10. Coley, W. B., and Hoguet, J. P.: Melanotic Cancer, with Report of Eighty Cases, Tr. Am. S. A. **34**:319, 1916.

11. Nitsch, Max: Neurofibromatose des Auges, Ztschr. f. Augenh. **69**:117 (Sept.) 1929.

12. Parsons, J. H.: Pathology of the Eye, London, Hodder & Stoughton, 1905, vol. 2, p. 518.

nevi as groups of densely packed chromatophores. Friedenwald [13] expressed a similar opinion.

Up to a few years ago most authors seemed to agree in admitting the connective tissue origin of chromatophores. Dr. Mary Knight,[14] in a study of melanotic neoplasms observed in the Mayo Clinic, expressed the opinion that melanin is produced only in ectodermal cells and that chromatophores are merely carriers of it. Broders and MacCarthy,[15] also of the Mayo Clinic, said positively that the pigment-bearing cells of the skin have their origin in ectoblastic rather than in mesoblastic tissue; they conceded that the origin of pigment cells in the eye is doubtful. None of these authors, however, has given any definite proof of his statements.

Regarding pigment in the uveal tract, distinction should be made between the construction of pigment and pigment-bearing cells. Miescher,[16] who did some excellent investigational work on this subject, found that pigment construction in the eye occurs only in the embryonic stage and that it is an oxidative process resulting from the action of an oxidative ferment, dopa-oxidase. The process of pigment construction is the same in the eye as in the skin. Proof of the presence of oxidase results from the dopa reaction,[17] which remains positive in pigment epithelium until the completion of pigmentation.

Oxidase occurs only in embryonic life; after the completion of pigment construction no more pigment is formed, and the cells concerned in pigment construction, whether melanoblasts or chromatophores, become, according to Miescher, only carriers of pigment during ordinary adult life. However, if there is any process of malignant degeneration the cells return to their original embryonic character and then have again the property of forming oxidase and resulting pigment. Under such circumstances, that is, in the presence of malignant degeneration in pigmented areas, the dopa reaction will again become positive. The results of the dopa reaction are, accordingly, an unmistakable proof that it is not a reaction of the pigment itself, as several authors have con-

13. Friedenwald, J. S.: Melanoma of the Choroid and Allied Tumors, in Penfield,[3b] vol. 3, p. 1065.

14. Knight, Mary S.: Melanotic Neoplasms of the Eye, J. A. M. A. **83**:1062 (Oct. 4) 1924.

15. Broders, A. C., and MacCarthy, W. C.: Melano-Epithelioma: Report of Seventy Cases, Surg., Gynec. & Obst. **23**:28, 1916.

16. Miescher, G.: Die Pigmentgenese im Auge nebst Bemerkungen über die Natur des Pigmentkorns, Arch. f. mikr. Anat. **97**:326, 1923.

17. Bloch, a distinguished dermatologist of Zurich, isolated a substance allied to tyrosine from the embryo of the broad bean. This substance (3, 4-dioxyphenylalanine) he called dopa. Its reaction is specific for all melanin-producing cells; these cells contain a ferment which converts dopa to melanin; the newly formed melanin colors the cell black.

sidered, but one that depends on the process of pigment building; the reaction takes place in the protoplasm of cells concerned in pigment construction.

Masson's [9a] assertion that in certain nevi the Schwann sheath cells can fabricate melanin has already been mentioned. In the histologic study of neurogenic pigmented choroidal tumors observed by them, Berger and Vaillancourt [18] extended Masson's conception; they noted that all or the greater part of the pigment cells in the eye as well as in the skin are of schwannian nature and that in neoplastic conditions melanogenesis can be assumed by neural elements, by the Schwann cells or by the syncytium of the Schwann sheath cells. This fact, according to these investigators, is absolutely incontestable. They observed that the tumor cells are capable of giving origin to both neural and pigment elements at once; these cells may differentiate on the one hand, as ganglion cells or typical Schwann cells, or, on the other hand, as pigmented cells free from all schwannian connections. This means, according to their theory, that pigment cells are nothing more than a particular differentiation of nerve elements; they also observed that this phenomenon of a double evolutive power of neoplastic cells in cases of melanotic tumors is not infrequent in cases of other neoplasms.

If these observations of Miescher,[16] Masson,[9] and Berger and Vaillancourt [18] regarding the origin of pigmented cells are correct, they will solve some of the puzzling questions that have arisen in the past regarding pigment and pigmented neoplasma. They are in harmony with the view of Knight [14] and others, who asserted that pigment is a product of ectodermal cells. Furthermore, Miescher's interesting disclosure that in the presence of malignant degeneration, sarcoma for instance, the normal cells revert to an embryonic state is in line with the view of Borst [19] and others, who have stated the opinion that sarcoma develops not from normal cells but from embryonal groups.

I must now return to the consideration of Masson's work concerning neurogenic neoplasma. In his second paper,[9b] published in 1932, he showed by histologic and biologic investigation of spontaneous and experimentally produced encapsulated nerves (including Recklinghausen's fibromatosis, schwannoma, neurinoma and all types of sensory and motor neuroma) that all such neural tumors have as their fundamental element proliferating cells of the nerve sheath of Schwann and are therefore ectodermal and do not originate from the mesodermal fibroblast. He showed that the collagen in these tumors is derived from the neurectoderm. He denied that the connective tissue of the endo-

18. Berger, L., and Vaillancourt: Ganglioneurome mélanique, Bull. Assoc. franç. p. l'étude du cancer **23**:275, 1934.

19. Borst, M.: Die Lehre von den Geschwülsten, Wiesbaden, J. F. Bergmann, 1902, p. 326.

neurium and perineurium has any part in the evolution of the neural tumor proper, though he stated that its proliferation may be stimulated by the adjacent tumor. Such growth of connective tissue may at times be so great as to stifle the Schwann cells. The syncytium of the Schwann sheath cells proliferates when from any cause the axon which it encloses has become separated from its trophic center and degenerates. Study of the syncytium of the Schwann sheath cells shows that they are capable under such circumstances of being transformed into bundles of aneuritic regenerated cells; when such bundles invade the ordinary connective tissue about them they are separated from it by endotheliform perineurium, formed in all probability not by the common connective tissue cells of the region but by cells emigrating from the endoneurium of the degenerated nerve.

The point is that a peripheral neural tumor develops independently of connective tissue, although proliferation of connective tissue in its vicinity may be stimulated. In cases in which the proliferation is extreme the Schwann sheath cells may be mistaken for connective tissue cells or for endoneural cells. However, Masson considered that there can be no doubt of their origin from neural elements.

In the United States Masson's histologic work has been confirmed by Foot,[20] Stewart and Copeland,[21] Geschickter [22] and others. Foot confirmed the schwannian origin of melanoma especially. Stewart and Copeland found that the fundamental cell of origin of so-called neurogenic sarcoma is the Schwann sheath cell. Geschickter showed that of nine hundred tumors of the peripheral nerves studied in the surgical pathologic laboratory of Johns Hopkins University eight hundred and fifty originated in the Schwann nerve sheath and that histologically there was a remarkable degree of uniformity in the majority of these tumors. He pointed out that proliferation of neurites does not take place unless Schwann sheath cells are present; such proliferation in the presence of ordinary connective tissue alone is highly hypothetic.

The work of the investigators cited has made it clear that the tumors described in the literature under the names of neurofibroma, plexiform or cirsoid neuroma, ganglionic neuroma, neurogenic sarcoma, nevi affecting nerves, melanoma and under other names are a closely related group allied to Recklinghausen's disease. They are neural neoplasms, the fundamental cell of origin of each being the Schwann sheath cell.

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20. Foot, N. C.: Concerning Histology of Melanoma, Am. J. Path. **8**:321 and 619, 1932.

21. Stewart, F. W., and Copeland, M. W.: Neurogenic Sarcoma, Am. J. Cancer **15**:1235, 1931.

22. Geschickter, C. F.: Tumors of the Peripheral Nerves, Am. J. Cancer **25**:377, 1935.

The histologic similarity between the neural malignant tumors (especially so-called neurogenic sarcoma) and malignant tumors of connective tissue has been responsible for much confusion. Geschickter and Copeland [23] dwelt on the close resemblance that neurogenic tumors bear to ordinary spindle cell tumors. Like fibrosarcoma, the neural tumor may have a marked fibrillary structure and under the microscope may show many spindle cells. Also, like the fibrous spindle cell tumors, which show a continuous gradation from the benign fibroma to the most malignant spindle cell form, neural tumors show a gradual transition from benign neuroma to the most malignant form, called neurogenic sarcoma. The pathologic distinction between tumors of the spindle cell series and tumors of the neurogenic series is not made easily. However, the existence of neural tumors as a distinct class would appear to warrant a new classification not based on cellular characteristics.

With this new orientation of neural tumors in mind I shall now discuss neural tumors of the choroid. What I desire to establish is that tumors described up to this time in the literature as sarcoma of the choroid are either pigmented or unpigmented neural neoplasms originating from Schwann sheath cells of the nerves that traverse the choroid. It will be necessary, therefore, to review the histologic features of these nerves.

NEUROLOGIC STRUCTURE OF THE CHOROID

Minot [24] in his "Human Embryology" (1892) stated that the choroid is of ectodermal origin. Later writers, however, have been almost unanimous in the opinion that the choroid is derived from mesoderm, and this is the view taken by Kolmer [25] and Berens [26] (1936) in recent textbooks.

For the purposes of this paper I am especially concerned with the neurologic structure of the choroid. Agababow [27] was one of the earliest (1893) to investigate and describe the nerves of the sclera and choroid. He showed that the short ciliary nerves form a plexus in the posterior part of the eye, especially on the choroidal surface. True ganglion cells are seen here, the plexus gangliosus ciliaris of Krause. The long posterior ciliary nerves have only a minor part in the building of this plexus.

23. Geschickter, C. F., and Copeland, M. M.: Tumors of Bone, ed. 2, New York, American Journal of Cancer, 1936, p. 591.

24. Minot, C. S.: Human Embryology. New York, William Wood & Company, 1892, p. 710.

25. Kolmer, W., in von Möllendorff, W.: Handbuch der mikroskopischen Anatomie des Menschen, Berlin, Julius Springer, 1936, vol. 3, p. 782.

26. Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936.

27. Agababow, A.: Ueber die Nerven der Sklera, Arch. f. mikr. Anat. **63**: 701, 1904; Ueber die Nerven in den Augenhäuten, Arch. f. Ophth. **83**:317, 1912.

Agababow further believed that the whole posterior half of the choroid to the exit of the venae vorticosae was traversed by ciliary nerves which sent fine branches to the suprachoroidal plexus and in which true ganglion cells were noted. He observed motor and vasomotor nerves in the choroid. The sensory nerve endings were in the sclera; none were seen in the choroid; however, he observed something analogous in the nerve network of the lamina suprachoroidea. Bietti's [28] investigations (1897) pointed to the conclusion that the nerves of the choroid have principally a vasomotor function.

Many later writers denied the occurrence of sensory fibers in the choroid.

In Salzmann's [29] "Anatomy and Histology of the Human Eyeball" (1912) it was stated that numerous nerve fibers, the last branches of the ganglionated plexus beginning in the suprachoroidea, traverse the choroidal stroma. In "The American Encyclopedia and Dictionary of Ophthalmology" [30] it was stated that many small branches derived from the ciliary nerves form a plexus in the lamina vascularis on their way through the suprachoroidea. They are provided with numerous large and multipolar ganglion cells showing origin from the sympathetic nervous system. Axenfeld [31] observed that the ciliary nerves pass through the sclera from the choroid in the anterior part of the globe. Nitsch [11] of the Vienna Eye Clinic said that sympathetic nerves and cell plexuses are normally present in the outside layer of the choroid. Berens [26] (1936) expressed the view that the long posterior ciliary nerves convey sympathetic fibers as well as sensory fibers. Berger and Vaillancourt [18] stated that Testut (1905) described and figured ganglion cells in the normal choroid and that they themselves observed such cells in the histologic investigation of the choroidal tumor in the case observed by them and were satisfied that they were of sympathetic origin. They expressed the opinion that the formation of sensory corpuscles is not confined to the central nervous system. All their investigations tended to show these writers that the ganglion cells form a peripheric neuron which is in connection with fibers coming from the ciliary nerves and that the choroidal tumor investigated by them originated from these ganglion cells.

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28. Bietti, A.: Sulla distribuzione e terminazione delle fibre nervose nel corpo ciliare, Ann. di ottal. **26**:215, 1897; Anatomische Untersuchungen über die Regeneration der Ciliarnerven nach der Neurotonia optico-ciliaris beim Menschen, Arch. f. Ophth. **49**:190, 1900.

29. Salzmann, M.: Anatomy and Histology of the Human Eyeball, translated by E. V. P. Brown, Chicago, University of Chicago Press, 1912, p. 56.

30. Wood, Casey, A.: The American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1914, vol. 3, p. 2161.

31. Axenfeld, T.: Nachweis und Bedeutung meiner "intraskleralen Ziliarnervenschleifen" am lebenden Merschenauge, Klin. Monatsbl. f. Augenh. **75**:602, 1925.

In connection with this question of sensory nerve fibers in the choroid, an interestnig observation has been made by Sédan.[32] This writer stated that in three patients with choroidal tumor whom he observed there was partial and limited anesthesia of the cornea in its external half, corresponding to the site of the lesion. The explanation, he thought, may be the fact that the tumor constricts the nerves in the suprachoroidal space by pressing them against the sclera and thus sensation is interrupted.

I have dwelt on the neurologic structure of the choroid because in connection with my own special work it is necessary to show that this vascular membrane is traversed by nerve elements, particularly the ciliary nerves, and that a neural neoplasm can easily arise in it, that is to say, that an ectodermal tumor can originate and proliferate in the midst of mesodermal tissue. As a matter of fact, a neoplasm containing neural elements is frequently observed in the uveal tract. Accordingly, I shall next deal especially with such neoplasms occurring in the choroid.

NEURAL TUMORS OF THE CHOROID

The most commonly reported tumors in the choroid are melanoma and so-called sarcoma. From what has been said earlier in this paper, melanoma, or melanotic sarcoma, may now be looked on as a neural tumor. Friedenwald [13] said that this tumor occurs in the choroid ten times as frequently as in the iris or the ciliary body. Miescher's [16] histologic investigations satisfied him that melanoma of the choroid originates in the choroid and not in the retina. Benign pigmented tumor of the choroid is rare, and generally the tumor becomes highly malignant.

Sarcoma of the choroid is perhaps the most important pathologic condition of the uveal tract. Annen [33] has classed it as pure melanosarcoma, pure leukosarcoma and mixed forms. Callender,[34] in an effort to arrive at a conclusion regarding its malignancy, classified so-called sarcoma according to cellular structure and cellular arrangement. According to "The American Encyclopedia and Dictionary of Ophthalmology," [35] sarcoma of the choroid is the chief intra-ocular growth of adult life, and its relative frequency in various clinics is from 0.03 to 0.06 per cent of that of all pathologic conditions of the eye.

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32. Sédan, J.: Anesthésies cornéennes dans les tumeurs de la choroïde, Bull. Soc. d'opht. de Paris, December 1934, p. 620; Arch. d'opht. **52**:595, 1935; Rev. gen. d'opht. **55**:167, 1936.

33. Annen, E.: Sarcomes choroïdienes, Ann. d'ocul. **170**:651, 1933.

34. Callender, G. R.: Malignant Melanotic Tumor of the Eye, Tr. Am. Acad. Ophth. **36**:131, 1931.

35. Wood, Casey A.: The American Encyclopedia and Dictionary of Ophthalmology, 1914, vol. 3, p. 2173.

The literature shows that the origin of sarcoma of the bulb and its pathologico-anatomic nature are only partly understood, though, as Nitsch,[11] Schubert [36] and other writers suggested, this growth is probably due to malignant degeneration of neurofibroma. Nitsch remarked that sarcoma of the choroid always originates in the outside portion of the choroid and gives as the reason the fact that the nerves, for the most part, lie there.

In 1877 Knies [37] made a report of the histologic study in sixteen cases of sarcoma of the choroid. In all these cases he observed that the tumor had its origin in the posterior section of the bulb and, more precisely, in the middle layers of the choroid. He cited Wecker as asserting that melanosarcoma most frequently arises in the anterior portion of the bulb, anterior to the equator. Wintersteiner [38] in 1900 reported a case of sarcoma of the choroid originating in Haller's layer, that is, the layer of large vessels. Schubert [36] in 1925 reported a case of leukosarcoma which he thought could be classified as an instance of tumor due to malignant degeneration of neurofibroma. I cite this case because the tumor seemed to Schubert to be a neurogenous growth the origin of which was in the long posterior ciliary nerve.

Reference to the possibility of sarcoma arising from a nevus was made in Parsons' [12] "Pathology of the Eye" (1905). Schieck's [39] pathologic and histologic description in 1906 of a sarcomatous degenerating nevus of the right iris is interesting, as it shows that at this early date pathologists recognized that sarcoma can develop from a nevus. This was, of course, prior to Masson's [9a] observation that nevi have a neural origin. In 1928 Wätzold and Gyotoku [40] published a histologic study of twenty-seven cases of sarcoma of the choroid, in only five of which the tumor originated in the ciliary body and extended into the choroid. The significant point is this: These investigators considered that choroidal sarcoma, owing to the alveolar structure and polymorphous cellular forms, suggests an ectodermal origin which, however, they concluded can be successfully established only if cellular elements of like derivation are proved to be present in the choroid. It is now known that such neural elements do occur in the choroid. Another inter-

36. Schubert, F.: Operation eines "Leukosarcoms" der Choroidea mit Erhaltung des Auges, Ztschr. f. Augenh. **56**:252, 1925.

37. Knies, M.: Sixteen Cases of Sarcoma of the Choroid, Arch Ophth. & Otol. **6**:376, 1877.

38. Wintersteiner, H.: Beobachtungen und Untersuchungen über den Naevus und das Sarkom der Conjunctiva, Ber. u. d. Versamml. d. ophth. Gesellsch. **27**: 253, 1899.

39. Schieck, F.: Das Melanosarkom als einzige Sarkomform des Uvealtraktus, Wiesbaden, J. F. Bergmann, 1906, case 24.

40. Wätzold, P., and Gyotoku, K.: Zur Pathogenese des Aderhautsarkoms, Arch. f. Ophth. **120**:209, 1928.

esting item in this report is the fact that only one of the twenty-seven growths was a leukosarcoma, the other twenty-six being pigmented tumors.

Cases of pure neuroma, sometimes termed neurofibromatosis, pigmented or unpigmented, of the choroid, have been reported. Snell and Collins [41] (1903) are credited as being the first to show that the choroid can be the site of tumors in neurofibromatosis of the ciliary nerves, and they described a plexiform neuroma in which the ciliary nerves were thickened by what at that time they considered as perineural and endoneural proliferation of connective tissue; the thickening of the choroid was also considered as showing that the ciliary nerves could be attacked by neurofibromatosis. Other cases of ocular neurofibromatosis have since been reported, by Sutherland and Mayou [42] in 1907, Murakami [43] in 1913, and Kyrieleis [44] in 1928.

Fuchs [45] observed two neuromas of the ciliary nerves composed almost exclusively of medullated nerve fibers. In one case there was a knotlike thickening of one of the ciliary nerves lying in the choroid.

In 1925 Dr. Mary Knight,[46] of the Mayo Foundation, reported a melanotic tumor of the choroid; the article had excellent illustrations showing the histologic observations. The thickening of the choroid was due, in the author's opinion, to excess of connective tissue. She considered the case as one of fibroblastoma or meningoblastoma. Her investigations convinced her that the chromatophores of the choroid do not produce pigment but are merely carriers of it. This would accord with Miescher's [16] observations, already alluded to, except that Miescher believed that under neoplastic conditions the chromatophores can revert to their original embryonic function and again produce pigment.

Berger and Vaillancourt [18] in 1934 reported a case of tumor of the posterior segment of the choroid. The tumor originated intrachoroidally. After an elaborate histologic study the authors arrived at the conclusion that the tumor was a melanogenous ganglioneuroma with sensory corpuscles, and they considered it to correspond with Masson's [9a] neuronevus of the skin. From a study of their histologic descriptions and

41. Snell, S., and Collins, E. T.: Plexiform Neuroma of Temporal Region, Orbit, Eyeball and Eyelid, Tr. Ophth. Soc. U. Kingdom **23**:157, 1903.

42. Sutherland, G., and Mayou, M.: Neurofibromatosis of the Fifth Nerve with Buphthalmus, Tr. Ophth. Soc. U. Kingdom **27**:179, 1907.

43. Murakami, S.: Zur pathologischen Anatomie und Pathogenese des Buphthalmus bei Neurofibromatosis, Klin. Monatsbl. f. Augenh. **16**:514, 1913.

44. Kyrieleis, W.: Ein Neurinom am Limbus corneae, Arch. f. Ophth. **119**: 119, 1928.

45. Fuchs, E.: Ueber schaumige Einlagerungen in der Aderhaut, Arch. f. Ophth. **118**:697, 1927.

46. Knight, Mary S.: A Critical Survey of Neoplasms of the Choroid, Am. J. Ophth. **8**:791, 1925.

illustrations Berger and Vaillancourt considered that the choroidal tumors reported by Snell and Collins [41] and by Knight [14] were counterparts of the tumor observed in their case; they found no reports in the literature of others of exactly the same kind.

With the exception of Berger and Vaillancourt's neuronevus of the choroid, none of the authors cited referred to or gave histologic evidence of the origin of choroidal neoplasms by proliferation of the Schwann sheath cells.

Dr. Algernon B. Reese loaned a section of an eye in which a nevus of the choroid and long posterior ciliary nerve was found on routine examination (fig. 1 *A* and *B*). The nevus was in no way responsible for the enucleation and on account of its location was not suspected.

The section was stained with hematoxylin and eosin. The eye had been cut in an almost true horizontal plane, the sections showing segments of the long posterior ciliary nerves on both the temporal and the nasal side. On one side the long posterior ciliary nerve was normal and as it neared the choroid was accompanied by only a few chromatophores. On the opposite side the nerve channel ran through the sclera in a long diagonal course. In its proximal part the channel contained normal-appearing nerve and part of a vascular wall accompanied by a few pigmented cells. The greater part of the canal was occupied by a nevus which widened the canal and ended in a nodule of heavily pigmented cells in the choroid, where these pigmented cells continued anteriorly for a short distance. Inside the scleral channel the cells, for the most part, were parallel with it, and as they reached the choroid they swirled about, assuming a position parallel to it. The nevus was composed of various cells: Long pigmented cells with faint nuclei strung along chiefly in the peripheral part of the canal but were seen throughout the nevus; there were long, blue-staining spindle cells with thin nuclei, and short, blunt spindle cells with large nuclei; the round pigmented cells varied in size from small to very large. The nodule in the choroid was so dense that the individual cells could not be recognized, but outlines of round, spindle and branched pigmented cells coulld be detected.

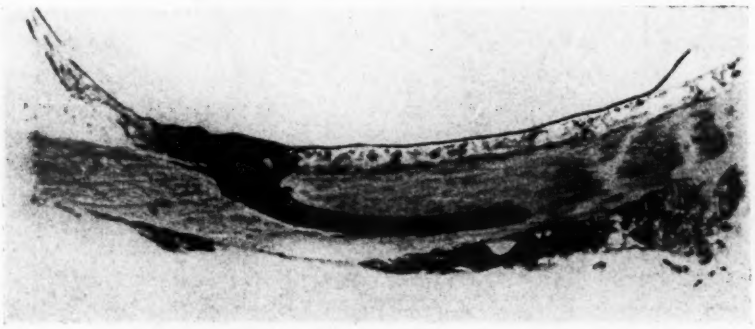
Occurring in the path of this large nerve—and recalling the observations of Masson,[9a] Foot [20] and others—this nevus, it can be said, undoubtedly arises from the Schwann sheath cells and the pigmented cells which normally may outline the ciliary nerves and vessels. It is possible that a malignant tumor may arise from just such a nevus of the ciliary nerve and choroid.

The structure and arrangement of the cells in uveal sarcoma have been described well in past literature on the eye. The derivation of the spindle cells in fascicular and palisade formations of the more anaplastic pleomorphous epithelioid type of cells and of the various types of pigment cells has been stated to be mesoderm.

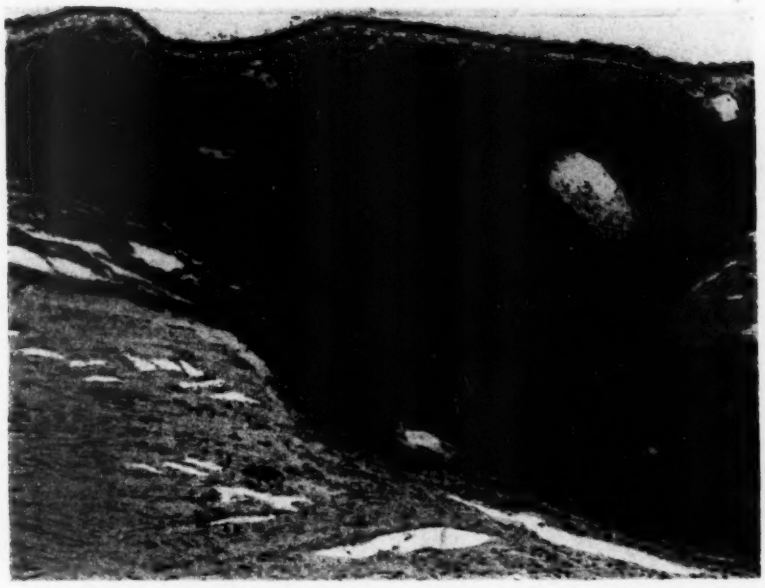
So far as I know, the specimens which I am about to describe are the first in which histologic evidence is furnished of the neural origin of so-called sarcoma of the choroid, that is to say, its origin from proliferating Schwann sheath cells of the ciliary nerves traversing the choroid.

PERSONAL WORK ON SARCOMA OF THE CHOROID

Dr. Richard Jaffé, of Chicago, after reading the work of P. Masson [9] concerning the neural origin of certain types of sarcoma, suggested that I search for nerves in relation with sarcoma in the choroid, where this



A



B

Fig. 1.—*A*, nevus of long posterior ciliary nerve and choroid. *B*, high power photomicrograph of the nevus.

type of tumor is frequent. At that time (1932) I had sectioned serially and stained every section of a bulb specimen (7) in the horizontal meridian, for the study of its normal anterior segment.[47] To the

47. Dvorak-Theobald, G.: Schlemm's Canal: Its Anastomosis and Anatomic Relations, Tr. Am. Ophth. Soc. **32**:574, 1934.

temporal side of the optic nerve, in the choroid, a tumor was observed, composed mostly of nonpigmented spindle cells; pigmented cells were seen in the periphery of the tumor and were sparsely present between bundles of cells. In this tumor, through sixty sections—almost 1 mm.—the long posterior ciliary nerve was seen intimately involved in the growth. It entered the tumor with well defined boundaries; it seemed to burst and bloom by proliferation of its Schwann sheath cells to form the surrounding tumor, and the nerve then continued forward on its accustomed way. The tumor was composed of spindle cells in bundle and sheet formation and was classified as a leukosarcoma.

Since that time a number of other bulbs enucleated because of a choroidal tumor have been sent to the laboratory.

The most frequent site of a uveal tumor is to either side of the optic foramen in the horizontal meridian, that is to say, in the path of the long posterior ciliary nerves; when it is farther forward it is off the horizontal plane, in the upper or lower nasal or temporal quadrant. Trying to trace nerves into a more anterior tumor would be a tedious task, as it might arise from any of the numerous short ciliary nerves or branches of the long posterior ciliary nerves. In all, seven bulbs were selected, sectioned serially and stained with hematoxylin and eosin, Masson's trichrome stain, Mallory's stain for connective tissue, Van Gieson's stain, Held's stain and Foot's silver stain. Six bulbs were cut as horizontally as possible so as to obtain longitudinal sections of the long posterior ciliary nerves. In five of these bulbs the Schwann sheath cells of the long posterior ciliary nerves were observed to be proliferating to form the tumors. The sixth bulb showed no involvement of the long posterior ciliary nerve. A small tortuous ciliary nerve traversing the choroid between the optic nerve and the long ciliary nerve was involved in a nevus formation. Because several important sections were lost in sectioning this bulb, the exact relation of this nevus to the tumor cannot be definitely known.

The seventh bulb was cut diagonally because of a sarcoma in the posterior upper temporal quadrant. By accident sections were cut longitudinally through a small ciliary nerve which ran through fifteen sections. As this nerve neared the choroid its Schwann sheath cells became more numerous and stained more brilliantly than the cells of the normal nerve. At its very entrance into the choroid the Schwann sheath cells proliferated into the tumor. All these sections showed the axons to be few and scattered. Other ciliary nerves passed through the tumor without change.

According to the present day nomenclature, the tumors here described are classified as spindle cell sarcoma and leukosarcoma; melanosarcoma, and mixed cell sarcoma. It was observed that nonpigmented tumors had their origin from Schwann sheath cells of nerves

which were accompanied in the scleral channel by few or no pigmented cells and that the pigmented tumors originated from the pigmented cells surrounding the nerve, the peripheral Schwann sheath cells or those of the entire nerve. In the cases in which the last origin was noted the normal long posterior ciliary nerve on the opposite side was accompanied by pigmented cells.

These tumors were so-called sarcomas of the choroid of various cellular structure—spindle cell, round cell, melanotic cell and mixed cell—in varying stages of maturity, some having chiefly mature spindle cells and others pleomorphous anaplastic cells. In some cases the tumor began in the nerve as it passed through its scleral channel; in others the tumor originated from the nerve intrachoroidally.

The microscopic observations in the aforementioned cases are given in the following section. As fine descriptions of the structure and cytologic features of similar tumors are to be found in the literature, I

Data for Seven Cases of So-Called Sarcoma of the Choroid

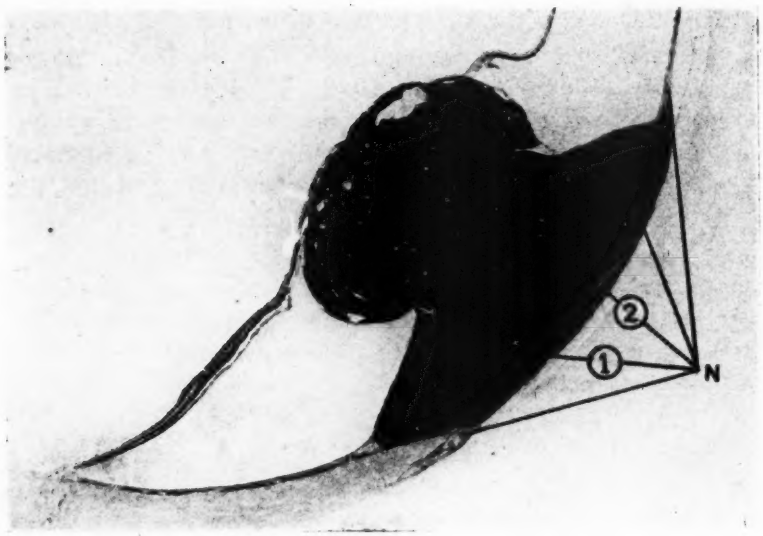
Date	Surgeon	Specimen	Name of Patient	Sex	Age, Years	Duration of Symptoms Prior to Operation	Pathologic Diagnosis
1932	Dr. Geo-ge Suker	1	.....	F	52	5 mos.	Spindle cell sarcoma
1/13/36	Dr. Harry Woodruff	2	F. E.	F	58	2 yrs.	Spindle cell sarcoma
1/17/35	Dr. T. D. Allen	3	D. S.	M	52	2 mos.	Melanosarcoma
7/19/35	Dr. Theodore Shapira	4	B. S.	M	32	?	Melanosarcoma
5/23/35	Dr. E. K. Findlay	5	R. M.	M	61	2 yrs.	Mixed cell sarcoma
7/12/35	Dr. T. D. Allen	6	E. J.	M	..	2 yrs.	Spindle cell sarcoma
7/—/—	Dr. R. J. Curdy	7	K. S.	M	..	.....	Spindle cell sarcoma

shall limit my own descriptions as much as possible to the relation of the nerves to the tumors. As the patient's history is not pertinent to the observations here described, only enough is given to identify the specimen; also description of the gross and microscopic appearance of the entire eyeball is omitted.

MICROSCOPIC OBSERVATIONS

SPECIMEN 1.—The pathologic condition in this eye depended entirely on the choroidal tumor, which had broken through the lamina basalis and was infiltrating the retina. The tumor was composed mostly of nonpigmented spindle cells; spindle cells were noted in its periphery and were sparsely present between bundles of cells. The tumor was slightly vascular, becoming more vascular after breaking into the retina, where the vessels were almost cavernous. Sections 340 to 400 showed interesting details concerning the relation of the long posterior ciliary nerve to the tumor.

As was mentioned previously, the sections were cut parallel with this nerve, and it could be traced through these sections from its entrance to its exit and beyond (fig. 2*A*). As the nerve entered the tumor its boundaries were well defined for a short distance. It widened; its boundaries broke, and the Schwann sheath cells proliferated, giving rise to the surrounding tumor (fig. 2*B*). The cells nearest the ciliary nerve were very mature, long spindle cells with pointed nuclei.



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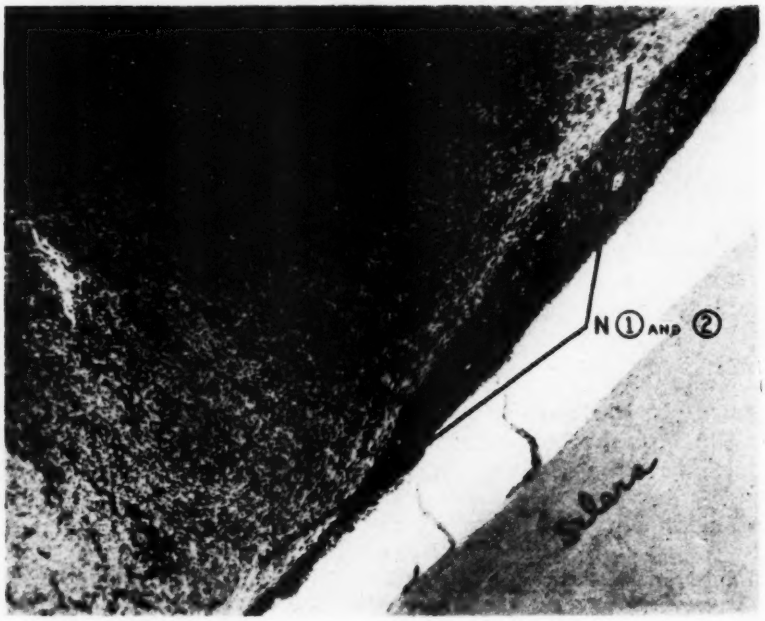


B

Fig. 2 (Specimen 1).—In *A*, *N* marks the course of the long posterior ciliary nerve. At *1* and *2* the Schwann sheath cells are proliferating to form the tumor. *B* shows the proliferation of Schwann sheath cells into sheets and bundles of tumor cells, growing almost at right angles to the nerve.

The cells grew into intertwining sheets and bundles, which were almost at right angles to the course of the nerve. The newer cells were shorter. The oval nuclei had one or more nucleoli. Where the tumor had broken through the lamina basalis the cells were very anaplastic and mitosis was active. Pigment cells were seen in the periphery of the tumor and between the bundles of cells.

With Masson's trichrome stain the Schwann sheath cells and the tumor cells were colored red, as were all other known ectodermal tissues. The cornea and sclera and the connective tissue about and lining the capillaries and various septums stained blue. The collagen, Masson said, is not mesodermal, but is determined by the Schwann cells, just as basement membrane is determined by epithelium. Sections stained with other methods corroborated this differentiation of the tissues. No masses of new connective tissue or granulation tissue were observed.

Summary of Histologic Study.—The growth was a malignant, only slightly pigmented tumor, originating intrachoroidally from the Schwann sheath cells of a long posterior ciliary nerve. The tumor was what has been commonly described as spindle cell leukosarcoma.

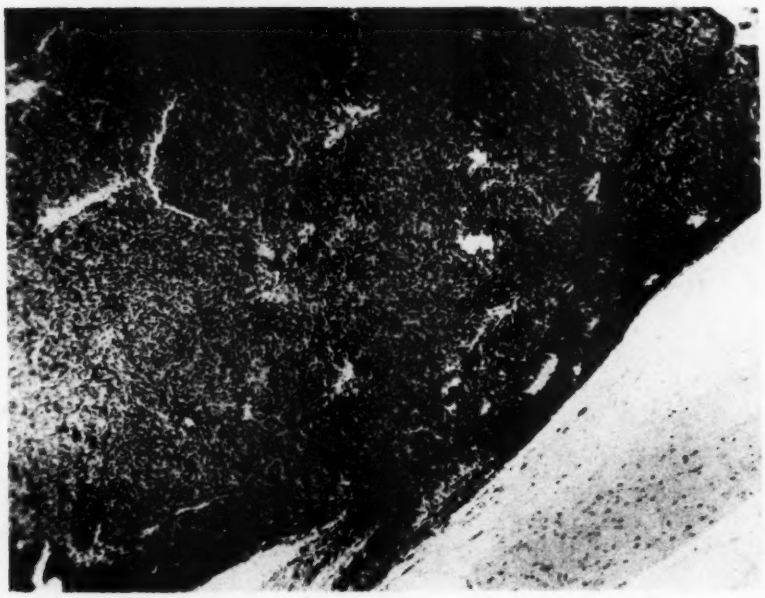
SPECIMEN 2.—This tumor was composed of large spindle cells with well defined nuclei and nucleoli. The cells grew in sheets and bundles which had only a small amount of collagenous material between them. Cells grew in whorls about the vessels, most of which were lined by collagen with endothelium. This was well brought out by the Foot silver stain. Pigmented cells in small numbers were seen near the sclera; they were mixed cells—spindle, round and chromatophore-like—of varying sizes and contained both fine and coarse granules of pigment.

Sections 290 and 381 passed through the long posterior ciliary nerve. As the nerve entered the scleral channel it was accompanied by a few pigmented cells (fig. 3*A*). Histologically, it appeared normal, with the axons, Schwann sheath cells and collagenous fibers in perfect order. After it wound over a larger artery its character changed; the nerve appeared in disorder, and the cells varied—there were long spindle cells with or without pigment; thick, blunt spindle cells with indefinite nuclei, like those found in nevi, and cells with well defined nuclei and nucleoli, like those found in the main body of the tumor. Winding through these cells were strands of normal Schwann sheath cells and capillaries. The axons, which are well seen in the normal nerve, were absent (shown by Held's stain). As the Schwann sheath cells progressed into the choroid, tumor cells with shorter and more blunt nuclei grew from them (fig. 3*B*). These cells in turn gave rise to others which were oval or even round.

With Masson's trichrome stain the normal Schwann sheath cells and the tumor cells took a red color; the axons stained red, and collagenous fibers stained blue. All the known ectodermal tissues stained red, and all the known mesodermal tissues stained blue. With Foot's silver stain only the collagenous fibers between bundles of cells and the outlines of vessels appeared pinkish brown; the tumor cells and the Schwann sheath cells did not stain. As serial sections of this tumor were studied the impression of bundles of Schwann sheath cells twining and intertwining became most apparent. As the process continued the cells became more anaplastic.

Summary of Histologic Studies.—This growth was a malignant, only slightly pigmented tumor originating from the Schwann sheath cells of a long posterior ciliary nerve and its accompanying pigment cells; the nerve was tumorous while still in the scleral channel. The tumor was what has been commonly described as spindle cell sarcoma.

A



B

Fig. 3 (specimen 2).—*A*, tumorous nerve traversing the scleral canal. *B*, higher magnification of *A*, showing pigmented cells in the nerve while still in the scleral canal.

SPECIMEN 3.—The tumor was composed of mixed cells. The largest part was composed of pigmented spindle cells of varying sizes; here the vessels were large and almost cavernous; some of these vessels were lined with endothelium, others with collagen. The smaller part of the tumor was composed of unpigmented cells and very slightly pigmented cells, more closely packed together in sheets and bundles.

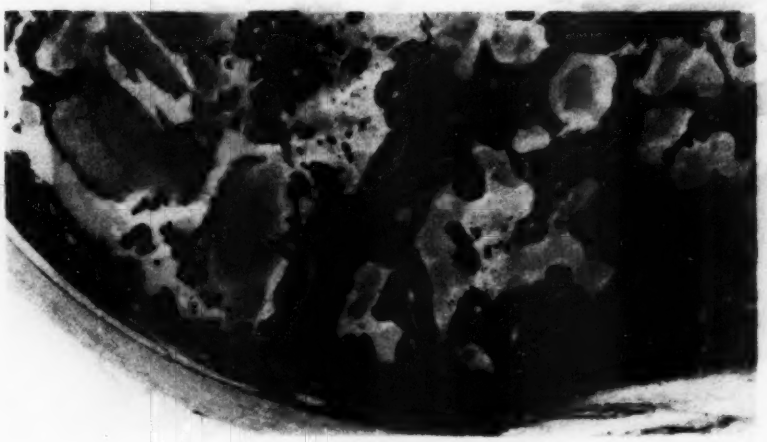
The long posterior ciliary nerve could be traced through sections 121 to 200. When the nerve entered the scleral channel it was accompanied by pigmented cells, which increased in number, size and content of pigment as the nerve neared the choroid (fig. 4). At this point the peripheral Schwann sheath cells began to proliferate into spindle cells which had more or less pigment. This could best be seen where the section had been slightly pulled in cutting and mounting. Large round cells with heavy pigment accompanied them; many of these cells had processes or feet that hugged the nerve. Farther on the most peripheral Schwann sheath cells and pigment cells proliferated to several layers; the unpigmented cells became blunt and rounded, and the chromatophores became round and had coarse granules, and their nuclei had one or more nucleoli. Division of cells was by amitosis. The nerve became thinner as it proceeded through the tumor and disappeared.

Comment.—In this specimen, in contrast to the two previously described, the perineural pigment cells, as well as the Schwann sheath cells, proliferated to form the tumor. These pigment cells, Miescher [16] showed, are also derived from neural ectoderm.

Summary of Histologic Study.—This growth was a malignant tumor of the choroid, originating in the Schwann sheath cells of a posterior ciliary nerve and its accompanying pigment cells. The tumor originated in the scleral channel. It was of the kind commonly described as melanosarcoma or mixed cell melanosarcoma.

SPECIMEN. 4.—The tumor was necrotic, and massive hemorrhages had occurred, probably after an operation for the relief of tension. Mixed cells comprised this tumor. Thin spindle cells became more blunt and wide where growth was rapid. There were large oval or round unpigmented cells with from one to three nucleoli in the large nucleus, which may be called epithelioid cells. Some of the spindle cells were pigmented. There were round pigmented cells of varying sizes with fine and coarse granules. The vessels were lined for the most part by collagenous fibers. There was a large extrabulbar extension between the sclera and Tenon's capsule, in which the nest of epithelioid cells were separated by wide collagenous bands.

The long posterior ciliary nerve was studied in relation to the tumor in sections 260 to 360. This nerve was of interest while still in its scleral channel (fig. 5 *A*). It was wide, and there was a marked decrease in the axons. The Schwann sheath cells were increased in number and irregular in arrangement. The external layers of these cells were proliferating and mingling with pigment cells which were massed about the nerve. As the nerve progressed into the tumor it became thinner, the Schwann sheath cells were more regular, and occasional segments of axons were seen. From the inner surface of the nerve the Schwann sheath cells and pigmented cells proliferated into sheets and bundles (fig. 5 *B*). These cells were blunt spindle cells which became more mature or anaplastic as the tumor progressed away from the nerve. With Masson's trichrome stain the Schwann sheath cells, as well as the tumor cells, stained red— the protoplasm a light red, the cellular and nuclear walls and the nucleoli a dark red. Foot's



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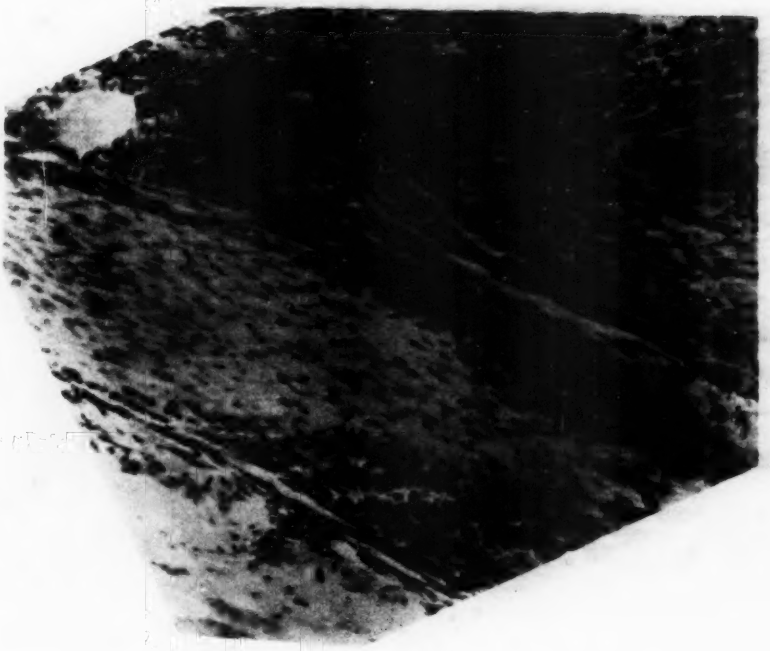


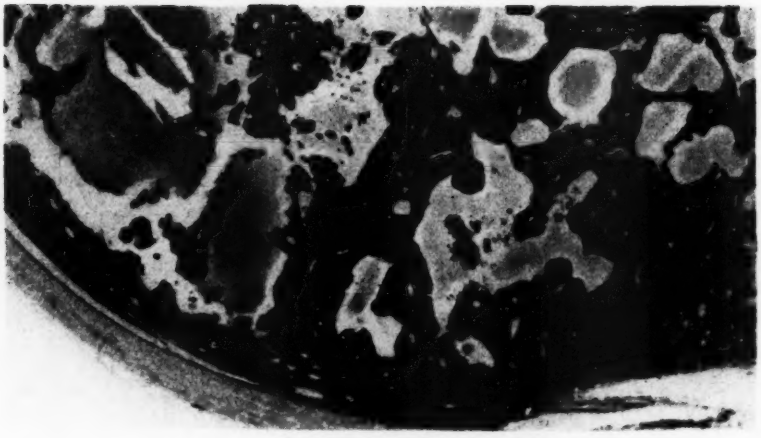
B

Fig. 4 (Specimen 3).—*A*, long posterior ciliary nerve entering tumor. *B*, detail of *A*, showing proliferation of tumor cells from the Schwann sheath cells and accompanying pigment cells.

A

B

Fig. 5 (Specimen 4).—In *A* the long posterior nerve is tumorous while still in the scleral canal. *B*, tumorous nerve showing pigment cells and proliferation of its peripheral cells into tumor cells.



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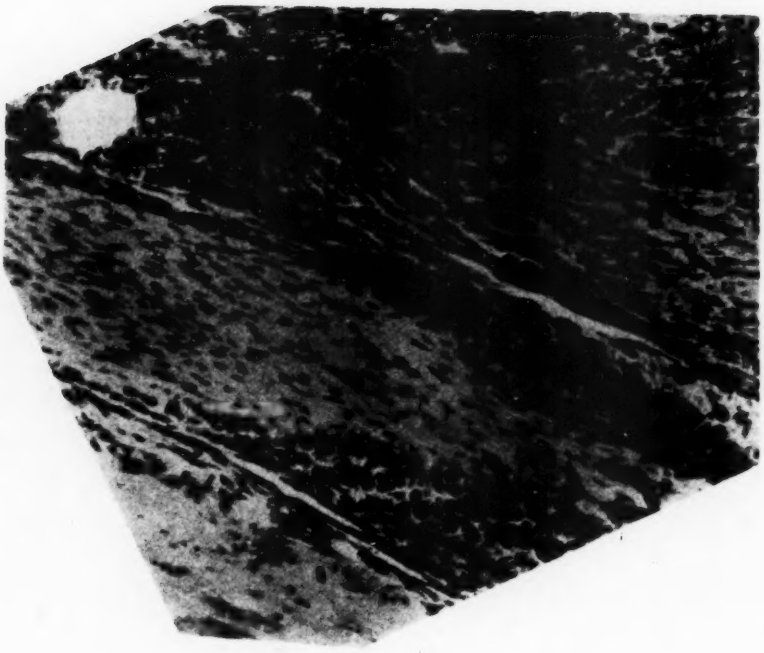


B

Fig. 4 (Specimen 3).—*A*, long posterior ciliary nerve entering tumor. *B*, detail of *A*, showing proliferation of tumor cells from the Schwann sheath cells and accompanying pigment cells.

A

B

Fig. 5 (Specimen 4).—In *A* the long posterior nerve is tumorous while still in the scleral canal. *B*, tumorous nerve showing pigment cells and proliferation of its peripheral cells into tumor cells.

silver stain silhouetted the collagenous fibers seen between cells and bundles of cells and lining the vessels. Held's stain was best for the axons. The long posterior ciliary nerve of the opposite side was normal and was accompanied through the scleral channel by pigmented cells.

Summary of Histologic Study.—This growth was a malignant tumor of the choroid, originating in the Schwann sheath cells of a long posterior ciliary nerve and its accompanying pigment cells while it was still in the scleral channel. It was of the type commonly diagnosed as melanosarcoma.

SPECIMEN 5.—The choroid on each side was occupied by a tumor; in the sections studied these tumors had no connection with each other. There were metastatic growths in the vitreous and the sclera, so it was thought that perhaps a connection would be found in other sections or in the calottes. The greater part of the choroidal tumors was necrotic. The live cells were in the periphery of the tumor; they were large, polymorphous cells with large nuclei and one or more nucleoli. They divided by both mitosis and amitosis. From their outlines, the necrotic cells were judged to be more spindle shaped. On the temporal side the long posterior ciliary nerve was converted into tumor, only a few central normal Schwann sheath cells being recognizable (fig. 6*A*). On the nasal side the nerve passed through the tumor with its boundaries well intact and remained independent of the tumor (fig. 6*B*). The staining properties of the cells of this tumor were identical with those of the tumors already described.

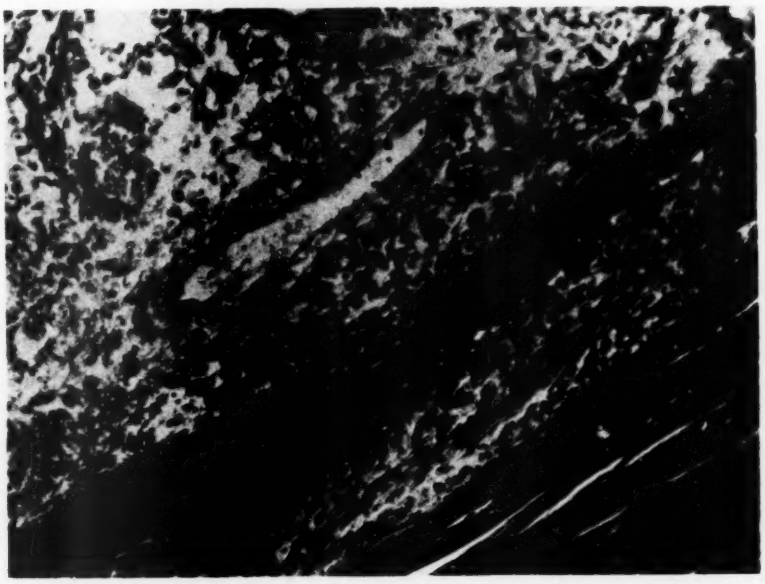
Summary of Histologic Study.—This growth was a malignant tumor of the choroid, originating in the Schwann sheath cells of a long posterior ciliary nerve and its accompanying pigment cells. It was of the type commonly diagnosed as mixed cell sarcoma.

SPECIMEN 6.—The tumor was a typical spindle cell sarcoma with fascicular cellular arrangement. The staining properties were identical with those of the previously described tumors. This eye was not cut truly horizontally, and tracing the nerves was unsatisfactory and difficult. The long posterior ciliary nerve, which was traced through sections 260 to 340, was found undisturbed by the surrounding tumor (fig. 7*A*). While still in the scleral channel its peripheral Schwann sheath cells proliferated into several layers (fig. 7*B*). Smaller nerves entered the tumor, accompanied by vessels, but their relation to the tumor could not be determined. A small tortuous ciliary nerve and its vessel, traversing the sclera between the optic nerve and the long posterior ciliary nerve, were involved in a nevus formation. The choroidal tumor may have had its origin in this nevus, but because several important sections were lost in cutting it cannot be definitely so stated.

Comment.—I am including this case to show that it is difficult at times to trace the nerves into the tumor. The nerve must be cut longitudinally and not on the bias. It is also entirely possible that many normal nerves course through the tumor, while the tumor originates from one.

Summary of Histologic Study.—This growth was a malignant tumor of the choroid, originating probably from the nevus in the sclera. This was a so-called spindle cell sarcoma.

SPECIMEN 7.—As was stated previously, this bulb was cut diagonally because the tumor was in the upper temporal quadrant in the posterior segment. The tumor was composed of spindle cells in fascicular arrangement. The cells nearest the sclera were long, slender spindles with dark nuclei, in which details could not be seen; these cells grew in sheets and bundles. As the tumor grew the cells



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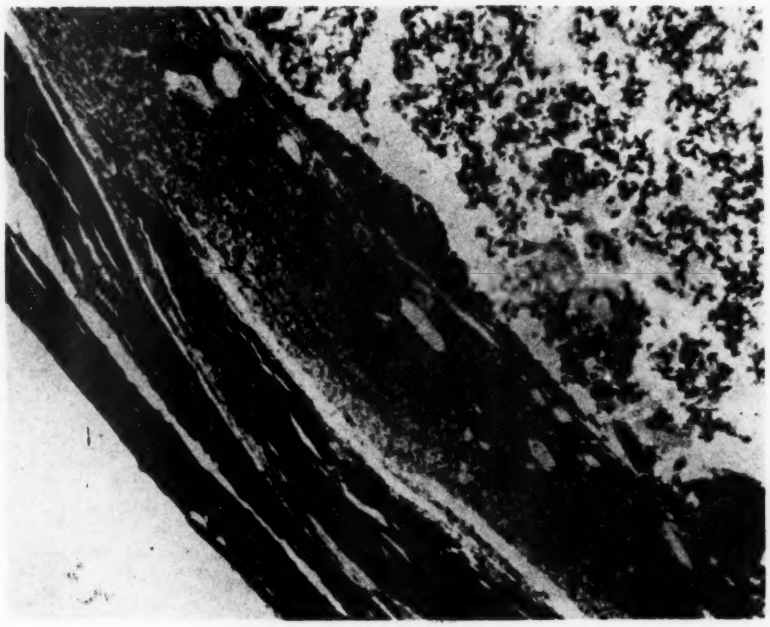
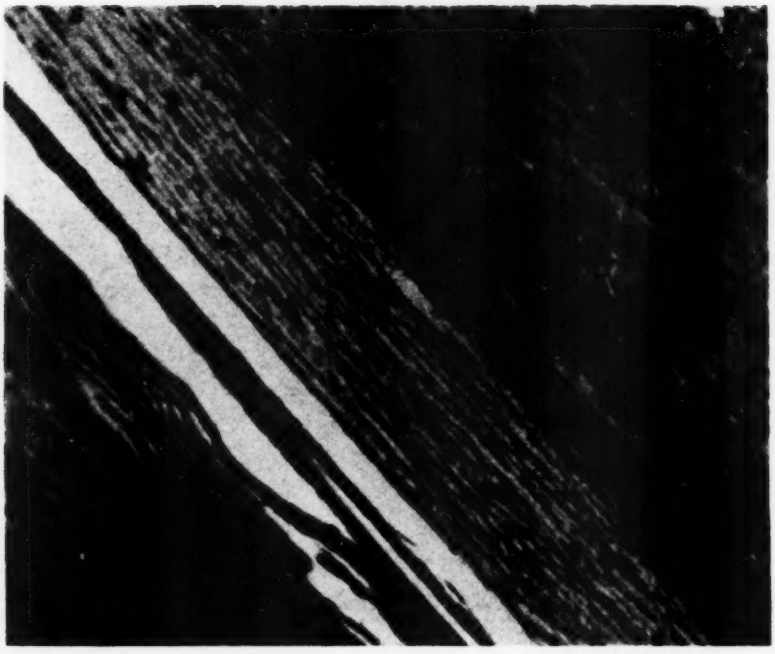


B

Fig. 6 (Specimen 5).— In *A* the long posterior ciliary nerve is shown converted into tumor, only a few central normal Schwann sheath cells being recognizable. In *B*, the nerve passes through the tumor with its boundaries well intact and remains independent of the tumor.

A



B

Fig. 7 (Specimen 6).— *A* shows the long posterior ciliary nerve traced through sections 260 and 340, undisturbed by the tumor. *B* shows that while still in the scleral channel the peripheral Schwann sheath cells of this nerve proliferated into tumor cells.

became shorter and the nuclei more blunt, and one or more nucleoli were seen. With further growth the cells became almost round and pleomorphous but were still in fascicular and palisade formations about vessels and spaces lined with collagen. Pigment cells were few, were present in the periphery of the tumor and had no part in its formation. The outside mature cells grew in sheets parallel to the surface of the tumor, forming a capsule for the fasciculated bundles of more anaplastic cells. This sheath broke anteriorly, and the new cells grew through the lamina basalis and invaded the retina. Sections 149 to 170 showed a short ciliary nerve coursing through the sclera in a long oblique path and entering the tumor (fig. 8 *A*). The nerve was accompanied by only a few pigment cells. Its Schwann sheath cells were normal in appearance and in number until the nerve neared the choroid. Here the Schwann sheath cells increased markedly

Fig. 8 (Specimen 7).—A short posterior ciliary nerve becomes tumorous as it nears the choroid, and gives rise to the tumor in it.

in number; they were long, thin spindle cells with heavily pigmented nuclei and continued on, blending with the tumor (fig. 8 *B*).

Summary of Histologic Study.—This growth was a malignant tumor of the choroid, originating in the Schwann sheath cells of a short ciliary nerve. It was a so-called spindle-cell sarcoma.

GENERAL COMMENT

Small choroidal sarcomas are disklike, and their peripheral cells form a laminated capsule. As the tumor grows the inner surface breaks and the tumor cells, being unconfined, grow rapidly into the characteristic mushroom shape. In the early stage sarcoma is much like the encapsulated peripheral nerve tumor the capsule of which is formed of

lamellae. Early authors recognized the fascicular type, which may show palisades, and a reticular type, and realized that despite these aspects the neurinomatous tissue is a unity. Masson [9b] showed that the palisaded formations in neurinoma are not accidental but are organoid products of sensory nerves, while nonpalisaded neurinomas are products possibly of motor nerves. The pathologic picture of various so-called choroidal sarcomas differs because of the fact that ciliary nerves carry both sensory fibers and motor fibers.

One can see by the study of these specimens that the syndrome of Sédan must occur not from pressure of the tumor on the nerve but by the interruption of the axons and the proliferation of the schwannian elements.

My observations that choroidal tumors originate from the Schwann sheath cell and the accompanying pigment cells is in general accord with the observations of Masson [9a] and of Berger and Vaillancourt, who identified pigment cells with Schwann cells. The finding corroborates that of Berger and Vaillancourt,[18] who said that it is an absolutely incontestable fact that in neoplastic conditions the nerve cells or the syncytium of the Schwann sheath cells can assume the property of melanogenesis. Nevertheless, the process of differentiation of pigmented neoplasms from unpigmented neoplasms under the circumstances would seem to call for further investigation.

I submit that I have proved the main point at issue, that is, the neural origin of so-called sarcoma of the choroid. The name sarcoma applied to neural tumors of the malignant type is a misnomer; it connotes connective tissue, and certainly these neural neoplasms are based on neither connective tissue nor epithelial tissue. Sarcoma was also distinguished by the spindle cell structure of tumors, but one sees that spindle cells may occur in tumors arising from neural ectodermal tissue as well as in those arising from mesodermal tissue. It is time to revise the classification of neoplasms, distinguishing them on the basis of embryonic tissue rather than on the theoretical basis of structure and cellular characteristics. As Broders and MacCarthy [15] justly observed: "Morphology is not an accurate criterion for embryologic origin in any specific embryonic layer. Moreover, the least differentiated tissues, such as the embryonic tissues, are more related to the tissues of tumors than to the tissues of normal organs."

SUMMARY AND CONCLUSIONS

The nature of neurogenic neoplasms in general is discussed, with particular reference to Masson's histologic investigations regarding cutaneous nevi and tumors of the peripheric nerves.

The literature regarding melanotic neoplasms and the origin of pigment is reviewed.

The neurologic structure of the choroid and neural tumors of the choroid, including those termed sarcoma, is described.

Original observations by myself in seven cases of so-called sarcoma of the choroid are given in detail, and from the histologic observations it is submitted that these tumors originated from the Schwann sheath cells of the posterior ciliary nerves in their passage through the choroid.

A revised classification of neoplasms is suggested, which would distinguish ectodermal neural tumors from those of other embryonal origin.

715 Lake Street, Oak Park, Ill.

Clinical Notes

DUGAS ON THE REMOVAL OF FOREIGN BODIES FROM THE EYE

CECILIA C. METTLER, A.M., AUGUSTA, GA.

Dr. Louis Alexander Dugas, M.D., LL.D. (1806-1884), held the chair of surgery at the Medical College of Augusta (the present University of Georgia School of Medicine) from 1855 to 1882, was dean for a period of twenty years and editor of the *Southern Medical and Surgical Journal* from 1851 to 1858. Eponymously he is remembered for his famous sign indicative of dislocation of the shoulder joint. During his professional career he published approximately one hundred articles which are a credit to his surgical versatility and the wide range of his medical knowledge. The paper which follows is one of several Dugas manuscripts which have recently come to light.

A SIMPLE PROCESS BY WHICH MOTES AND OTHER FOREIGN BODIES MAY BE REMOVED FROM THE EYE

BY LOUIS A. DUGAS

Read before the Augusta Library and Medical Society
December 15, 1876

The removal of motes or fragments of foreign bodies from the external Surface of the eye is an operation we are frequently called upon to perform.

Rail Road employes and travellers, workers in metals and Stone Cutters, are those most frequently claiming our Services. The natural Sensitiveness of the external eye is usually very much increased when we are consulted. So that the intolerance of light makes it difficult to examine the eye thoroughly.

With a little careful manipulation however we may succeed in finding the foreign body upon the cornea, the ocular conjunctiva or beneath the eyelids. Whenever found it is more or less difficult to remove by the procedures usually recommended by written authorities, and which you know consists simply in its removal by means of one or other form of instrument while the eyelids are held open. No directions are given for the purpose of rendering the eye motionless during the operation. And yet it is extremely difficult for the surgeon as well as painful to the patient to dislodge the foreign body while the eye is instinctively avoiding every approach of the instrument. In order to Surmount this difficulty I have for many years been in the habit of placing the end of the index finger upon the eye just within the canthas, and retaining it there until I have removed the object. The contact of the finger produces a sensation which, while not decidedly painful, is yet sufficiently decided to engross the attention of the patient and to prevent his moving the eye at the approach of the instrument or on its contact with the ocular surface.

From the Department of Anatomy, University of Georgia School of Medicine.

By this plan the foreign body may be removed from the surface of the eye as readily as from any other part and without the risk of Scratching or otherwise injuring the organ by repeated and unsuccessful attempts to take it by surprise, if I may use the expression, by sudden thrusts of the instrument used for the purpose. I am in the habit of using Scarpa's cataract needle, and find it better adapted to the purpose than any other instrument, whether the mote be embedded or in Simple contact.

A young man accompanied by his father came from one of our upper counties to get me to remove a thorn sticking in his cornea. It seems that he was walking in the garden and passing by a rose vine when a branch coming in contact with his eye one of the thorn plunged into the cornea and was left there by the onward movement of the young man. He applied to the physicians of the neighborhood, one after another who made unsuccessful attempts to remove it with pocket knives, bistouris, lancets, etc. When he arrived here I found him badly scarred and very despondent. By putting the end of my fingers upon the eye-ball so as to keep it quiet the thorn was at once removed without any difficulty whatever—I may add that the delighted father exclaimed: "How strange it is that no one else thought of this Simple method of proceeding."

I am not prepared to say that no else has ever resorted to this method, but I have been teaching its advantages very many years to the classes of the Medical College of Georgia.

It is more than probable, that other surgeons have used it, and, like myself, omitted to publish their views.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

SURGICAL TREATMENT OF STRABISMUS

REVIEW OF THE RECENT LITERATURE

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One hesitates to add to the great bulk of literature on the surgical treatment of strabismus. Yet it is the vast and ever increasing number of articles on this subject which has made it seem desirable to attempt a summary of the more recent reports so that the busy surgeon may have some idea of the present status of this important branch of ocular therapy. Practical considerations have made it necessary to limit this review to the literature of the last seven years (from 1930 through 1936). A conscientious attempt has been made to include everything written on the subject during this period, to point out the important contributions and to give the opinion of the majority wherever possible.

For the purpose of this review it has seemed expedient to divide the material into two groups: that on nonparalytic strabismus and that on paralytic strabismus. In the section on the surgical treatment of nonparalytic strabismus the various operations for weakening and strengthening the action of the ocular muscles are discussed. In the section on paralytic strabismus the operation of choice for each muscle is given.

NONPARALYTIC STRABISMUS

Complete Tenotomy.—This procedure has few exponents today. Carroll and Blake in their splendid research on rabbits pointed out that the results of tenotomy are unpredictable because of the failure of the muscle to become reattached to the globe in some instances. Peter considers complete tenotomy dangerous. In his detailed analysis Folk concluded that complete tenotomy in convergent strabismus is uncertain and leads to divergence, while in treating divergence there is no danger. The great uncertainty of the results and the occasional severe overcorrection have caused the majority of writers to abandon tenotomy in favor of one of the more controlled operations. However, some writers still advocate it. Hepburn has protested against the wholesale condemnation of tenotomy. He considers this operation very useful.

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especially for young children who require a general anesthesia. Bufill believes that simple tenotomy has often given excellent results. Cosgrove, Argañaraz and Perwög favor bilateral tenotomy of the internal rectus muscles in cases of severe strabismus. Onfray uses tenotomy only when there is no hope of binocular vision without it and then waits until the patient is 16 or 18 years of age before operating. Terrien, while admitting that this operation has been too extensively employed, stated the opinion that it should be retained, with the following reservations: It should be used only for strabismus of very high degrees; it should be limited to one muscle; the lateral attachments should be preserved; it should never be used before the patient is 10 or 12 years of age, and it should be used with an advancement, the aim being toward overcorrection.

Partial Tenotomy.—Opinions as to the value of partial tenotomy vary widely. Some writers (Peter, Folk) consider it practically useless, while others have advocated its use, usually in conjunction with one of the shortening operations (Burch and Grant, Castresana, Lordan, Hosford and Hicks, O'Connor). Stevens' central tenotomy is still being used by Hosford and Hicks and by Lordan. O'Connor described a two stage tenotomy for use with his cinch shortening operation. The first stage is central tenotomy. Six weeks later, after the central portion of the muscle has become reattached, he cuts the margins. He placed emphasis on maintaining control and avoiding torsion. Pollock has given the only description of tendon lengthening, performed by a modification of the method of Bishop Harman.

Various methods are in use for controlling the effect of tenotomy. While the details vary widely, the fundamental principle is to place a suture in the muscle or tendon before performing the tenotomy and then to secure it more or less firmly to either the stump (Thaw, Gorst) or the overlying conjunctiva (Alexiadès, Goldenburg). Usually the suture is tied lightly and left long for later adjustment. Folk considers guarded tenotomy less safe than partial tenotomy because of the danger of sutures slipping.

Recession.—The various types of tenotomies have largely been replaced by the recession (retroplacement) operation in recent years. Judging from the reports in the literature, the recession operation is the most popular operation on the ocular muscles today, used either alone or in conjunction with a shortening operation. The fundamental principle of this procedure is firm anchorage of the tenotomized tendon to the sclera at a measured distance behind the original insertion. It is to the splendid, painstaking work of Jameson that ophthalmologists are largely indebted for this principle and for the carefully detailed technic that has led to its wide acceptance. The critics of this operation

point out the danger of perforating the sclera with the needles, with resultant intra-ocular infection, and setting the muscle too far back, which causes crippling of convergence. That both mishaps can occur is undenied, but that they should not occur if the proper technic is followed is the claim of the many supporters of the operation. The long list of enthusiastic users of this procedure and the fact that intra-ocular inflammation has rarely resulted from its use bespeak its safety in a convincing fashion. While some writers (Gorst, Milner, Naftzger) have failed to note weakening of convergence after excessive recession, many more have seen such weakening, and the majority follow Jameson's carefully studied rule of not setting the insertion behind the equator. Opinion is not so uniform with regard to the external rectus muscle, the insertion of which is about only 2 mm. from the equator. Jameson is firm on this point, yet many feel that it is safe to transplant the external rectus muscle back 4 to 5 mm. While the indications for recession have been fully detailed by Jameson and by Berens, there is no uniformity of opinion on this point in the published reports. Some authors use this procedure as the operation of choice, regardless of the type of squint; many combine it regularly with one of the shortening operations, while still others use it only to weaken the action of a hypertensive muscle. Berens and Jameson believe that with greater experience with the recession operation one does fewer shortening operations. Several writers agree with Jameson's rule that 1 mm. of recession will correct 5 degrees of deviation. The amount of correction from one recession varies greatly (from 10 to 50 degrees). Jameson expects from 20 to 25 degrees of correction from a 5 mm. recession of the internal rectus muscle and from 40 to 50 degrees of correction from a bilateral recession. Recession of the external rectus muscle gives about one third of this effect. Jameson favors one or two recessions for convergent strabismus and reserves the addition of an advancement operation for squint of very high degree or in cases in which the operation must be confined to one eye. Jameson and others have not observed retraction of the caruncle after recession, while others have found this an annoying sequel. It may be important in this connection to note Jameson's technic of not undermining the caruncle. For the details of the technic of the recession operation the reader is referred to Jameson's articles. A number of innovations have been described (Berens, Bickerton, Curdy, F. A. Davis, Gifford, Gorst, Lancaster, Lombardo, Milner, Nugent, Peter, Prangen, Wilkinson), most of which consist of variations in the number and placement of the sutures without alteration of the fundamental principle or plan.

Tucking.—Burch and Grant are the chief exponents of the operation known as tucking, in the period under consideration. In their excellent

article giving their extensive experience with tucking they described their technic in detail and laid down definite rules for the amount of tuck on each muscle. The technic was also described by Speas, who in addition reported on a new instrument. That tucking is relatively safe no one will dispute. Its accuracy is questioned because of the incalculable amount of stretching. Experience seems to be the only remedy for this, which would make one doubt the validity of any figures. Necrosis of the muscle, slipping of sutures and an unsightly lump produced by the tuck in the muscle are other disadvantages reported. The conclusion of the advocates of tucking is that it is ideal for phorias or very small tropias (those up to 15 degrees). Beyond this it is ineffective and its safety would seem to be overbalanced by its disadvantages.

Advancement.—The question of the fate of the advanced portion of a tendon has not been settled. Some observers (Prangen, Horay) believe that the tendon or muscle becomes adherent to the stump, rendering everything in front of it useless. Jameson pointed out that the muscle fibers may function within an adherent capsule. Peter favors advancement over resection because from 5 to 10 degrees more effect can be obtained by including the conjunctiva, the capsule and the muscle in the shortening process; this operation produces less disturbance of the capsule than resection, and it is better to reattach by tendon than muscle. The objections to advancement other than the uselessness of the advanced portion are: the danger of scleral sutures, the difficulty of placing such sutures securely, the risk of torsion and the unsightly thickening that may persist. All but the last may be eliminated by proper technic. A number of surgeons (B. Moore, Onfray, Saint-Martin, Woodruff, Peter) use the advancement operation as the primary procedure in convergent strabismus, performing it on the external rectus muscle of each eye, if necessary. The great majority, however, use it for strabismus of low degrees to strengthen a hypotensive muscle, or for strabismus of higher degrees, in combination with a recession operation or some form of tenotomy. A few (Bickerton, Jameson) hold it in reserve for later operation if recession proves inadequate. Fifteen degrees is about the average correction from one advancement. Judging from the number of reports on this procedure, advancement is the most popular shortening operation today. The technic first described by Worth is the one most frequently followed. Pollock, Lagrange, Peter, Saint-Martin and others have reported variations. The most striking feature of many of the technics is the complexity of the sutures.

Resection.—The resection operation as described by Reese has served as the model from which most of the variations have branched

in this country. The recent literature on this procedure has conceded it a permanent place in the surgery of ocular muscles and has dealt largely with the details of the technic of the operation. The objections raised to the resection operation are chiefly the danger of sutures slipping and the failure to get sufficient effect. The large number of surgeons who have used this method with eminently satisfactory results indicates the rarity of slipping sutures. Failure to gain sufficient shortening is largely a matter of the technic of not placing the sutures sufficiently far back in the muscle, unless one agrees with Peter's dictum that the resection should not extend beyond the tendon. The question of anesthesia assumes importance here, as it is extremely difficult to obtain by local injection the complete anesthesia required for a large resection. The advocates of resection point out that, in addition to its comparative simplicity and the ease of reanchoring the muscle to its original insertion, it obviates largely the disadvantages of advancement, which have previously been mentioned. The placement of the sutures in the Reese operation causes the muscle to be folded under the stump, and the outer margin of the conjunctiva to be carried between the muscle and the stump. This unsurgical closure has given rise to a number of minor variations, two of which will be described briefly. After making a long curved incision near the outer canthus, Berens places the Prince forceps 1 mm. from the point of resection. He measures this with calipers, or with his new forceps on the shank of which is a millimeter scale. Two double-armed sutures are placed, one near each border, 5 mm. back of the forceps, from within. After the resection the sutures are passed through the stump and conjunctiva and tied. The conjunctiva is closed separately. Lancaster uses a longitudinal incision extending from the limbus to the canthus. The capsule is cut along each margin, and the Prince forceps is applied just in front of the point to be reattached. After the tenotomy two double-armed sutures are put through the stump from beneath and then through the muscle back of the forceps from within. The middle third of the muscle is not included in the sutures, to avoid strangulation. The eyeball is rotated toward the muscle, and the muscle is drawn forward with the Prince forceps by the assistant. Thus the sutures may be tied without tension. The excess part of the tendon is then excised, and the conjunctiva is closed with a running stitch.

Cinch Shortening.—In surveying the recent literature on the O'Connor cinch shortening operation one is impressed by the enthusiasm of almost all the surgeons who have reported on it. No other surgical procedure has called forth such extravagant statements from mature ocular surgeons of high standing, in this country, in recent years. The following statements may be cited as examples: "From the standpoint

of efficiency, safety, certainty of action and surgical principles upon which it is based, no operation yet devised can compare" (McCool); "the most perfect mechanical means of straightening an eye at any age" (H. Barkan). Some surgeons prefer it for the smaller deviations, but others believe that it will give as much shortening as the resection or the advancement operations. Peter found it particularly suited for shortening an internal rectus muscle because of the difficulty of doing other shortening operations in the limited space. The objections raised to the cinch shortening operation are the limited effect from the shortening and the marked reaction from the operation. For the exact technic and the arguments in favor of this operation the reader is referred to excellent descriptions, with illustrations, by Barkan, McCool, Hosford and Hicks, and O'Connor. Jackson found it to be the general verdict of all who have given the cinch shortening operation an intelligent and extended trial that it is superior to other shortening operations.

One of the newer and more original contributions to the surgical treatment of strabismus is the method for shortening a muscle by means of the myocampter, perfected by Barraquer. In recent years it has enjoyed considerable popularity in Spain and France. This is a completely bloodless procedure. The conjunctiva, Tenon's capsule and the muscle are grasped with a forceps 10 mm. from the insertion of the muscle. These structures are transferred to the hook of the myocampter and drawn into the instrument by a screw, in a fashion similar to that of most tuckers. The *agrafe,* a silver clip held in the jaws of the instrument, is pinched into position, clamping the entire fold of the conjunctiva, capsule and muscle. This is left in place from three to five weeks. It may fall away in ten days or may remain for six months or more. It is claimed that the *agrafe* is well tolerated. The advocates of this procedure emphasize its extreme simplicity and safety. The operation can always be repeated.

A number of original procedures have been described that may gain acceptance when they have had more thorough trials. Hubbard shortens a muscle by cutting it through well back and overlapping the two portions with mattress sutures. Dudinov's method for shortening a muscle resembles O'Connor's, auricular cartilage being used in place of the dermal sutures. The size of the cartilage controls the amount of correction. To decrease the effectiveness of a muscle, Chevasse has devised two methods. In his "posterior partial myotomy" he places a clamp as far back as possible, and with specially curved scissors he cuts into the muscle from each side, dividing up to four fifths of the fibers. In his "thermoplasty" he blocks off the nerve supply to the anterior segment of the muscle by a transverse band of electrocoagulation in the belly of the muscle. The portion of muscle in front of the band of coagulation becomes a fibrous band. Salvati advocated the

injection of absolute alcohol into the muscle. Dudinov tried this method in two cases and found that, in addition to great pain after the injections, the correction was lost in about three months. Smukler combines with a tuck of the external rectus muscle a stretching of the opposing internal rectus muscle. He accomplishes this by increasing the size of the tuck for three minutes. This apparently weakens or temporarily paralyzes the internal rectus muscle, allowing time for the tuck to adhere. He had uniformly splendid results in a large series of cases.

Operation of Choice.—To decide the operative procedure of choice from reports in the available literature is extremely difficult. One writer of vast experience may declare in favor of the recession operation as the primary procedure, reserving the shortening operation as an auxiliary. Another surgeon of equal standing may take exactly the opposite stand. Heated arguments on this question between ophthalmologists of experience are being carried on in the current periodicals. However, one finds that the majority have taken the middle course and have combined both principles, usually at the same operation and on one eye. The recession operation is the unquestioned favorite among the operations for weakening an ocular muscle; for strengthening a muscle there are several choices, each with some merits, but resection and advancement are most widely employed. It is striking how little real information can be gained from statistical reports of the results of surgical treatment of strabismus. A number of favorable analyses are found in the recent literature, but they are of scant assistance in the evaluation of the various procedures.

Age for Operation.—With the exception of a representative group of French oculists (Lenoir, Onfray), only a few surgeons (Hill and Courtney, Penman, Argañaraz, Georgariou) still favor waiting until near puberty before operating for strabismus. The great majority, however, favor operating as soon as more conservative methods of treatment have been given trial and failed, regardless of age. This means that many children are ready for operation at 3 years of age or earlier, and the consensus in this country is that the operation should be done at this time. Notable among the opinions of European authors is Sattler's emphasis on this point. Dunnington urged early operation to avoid secondary muscular changes, postoperative diplopia and the great physical and mental handicap.

Anesthesia.—The question of the type of anesthesia cannot be separated from that of the proper age for operation. Many English surgeons wait deliberately until local anesthesia can be used so that the effect of the surgical procedure can be determined during the operation by the use of the Maddox rod and graded accordingly. In this country it is generally believed that the advantages of early operation

greatly outweigh this, and general anesthesia has been used increasingly, with satisfactory results. In discussing this subject Dunnington stated that general anesthesia is no handicap if a careful preoperative diagnosis is made. If the patient to be operated on is an adult, there is greater variation of opinion. Some surgeons prefer general anesthesia at any age because of the belief that complete anesthesia by local means is rarely achieved, making a generous resection or advancement difficult or impossible. The successful use of tribromethanol in amylene hydrate has removed most of the objections to a general anesthetic, but this should be combined with a local anesthetic or with small amounts of ether by inhalation. Of the methods for administering a local anesthetic, in addition to the usual instillation of cocaine or one of the newer substitutes, such as butyn, injections of procaine hydrochloride into the muscle or within the muscle cone have met with most favor.

Sutures.—Silk sutures are still being used by the majority of surgeons. However, recent improvements in catgut sutures and particularly in the needles and the method of attaching them to the sutures have caused a number of surgeons to adopt such sutures for routine use. The great advantage of not having to remove the sutures has definitely outweighed the increased cost and the somewhat greater inconvenience in handling them. The sutures of 0000 ten day chromic catgut with atraumatic needles have been established as very satisfactory for work on the ocular muscles.

The great majority of writers on the surgical treatment of squint are on record as favoring conservative treatment before resorting to operation. This consists of full correction of refractive errors as measured after cycloplegia has been induced, correction of amblyopia by various forms of occlusion, and stimulation of fusion. On the last point, however, most of the reports are exceedingly vague. Comparatively few authors have advocated postoperative exercises. The recommendations on this point have been more specific. The stereoscope is the most popular instrument for postoperative treatment, and exercises are usually begun as soon as the bandages are removed. Jameson opposes postoperative exercises as unnecessary and capable of doing harm to a wounded muscle, possibly inducing external deviation through stimulation of the opposing muscle.

PARALYTIC STRABISMUS

Paralysis of the external rectus muscle is treated by means of transplantations of the tendon by a number of authors. Gifford, Beliaev, Goodman, Lancaster and others have described modifications of the Hummelsheim procedure. The principle is that of dividing the superior and the inferior rectus muscle longitudinally and transplanting the

outer portions (one third or one half) to the tendon of the external rectus muscle. The external rectus muscle may be advanced at the same time. Good functional results from this operation have been described. O'Connor utilizes the same principle but claims that he gets greater abduction by transplanting the halves of the vertical tendons farthest from the paralyzed muscle. Wiener confirmed O'Connor's results but described a new technic for obtaining the same effect in a simpler manner and without injuring the vertical rectus muscles. The external rectus muscle is cut off well back (15 mm.), and the anterior portion, still attached to the globe, is split longitudinally into two halves. The upper half is attached to the tendon of the superior rectus muscle and the lower half to the inferior rectus muscle. White pointed out that the transplantation operations are usually accompanied by recession or tenotomy of the internal rectus muscle, which may be a large factor in the result obtained. He is opposed to using the vertical rectus muscles because of the danger of adding a vertical deviation. He favors advancement of the affected muscle with recession of the direct antagonist. If there is a marked secondary deviation he does a recession of the internal rectus muscle of the other eye. For congenital bilateral paralysis of the abducens nerve Chevasse advised bilateral retroplacement aiming at fixed parallelism.

Little has appeared in the literature on the surgical treatment of paralysis of the internal rectus muscle. The most widely accepted treatment for this condition is shortening of the paralyzed muscle. In a case in which there was paralysis of the internal and the inferior rectus muscle and the inferior oblique muscle after a stab wound Peter was able, after recession of the external rectus muscle and tucking of the internal rectus muscle, to obtain good lateral rotation by transplanting the superior oblique muscle. Through an incision in the upper lid he cut the superior oblique muscle from the globe, opened the trochlea, brought the tendon down through a hole in Tenon's capsule and sutured it to the insertion of the internal rectus muscle. The excess part of the tendon was cut off. This procedure is not applicable to an adult with good vision in each eye. Wiener also reported transplanting the superior oblique muscle to the insertion of the internal rectus muscle. In secondary divergence after complete tenotomy of the internal rectus muscle some surgeons have had difficulty in finding the tenotomized muscle. In these cases Bielschowsky, La Rocca and others obtain a cosmetic correction by advancing the capsule, while Jackson has advised transplanting parts of the superior and the inferior rectus muscle.

For paralysis of the superior rectus muscle, shortening the paralyzed muscle or myectomy of the inferior oblique muscle of the other eye was recommended by White and by Dunnington. The choice of operation is determined largely by the amount of overaction of the inferior oblique

muscle and by which eye is used for fixation. The danger of ptosis and of diplopia in the lower field, after shortening of the superior rectus muscle, tips the balance in favor of myectomy of the inferior oblique muscle of the other eye. The myectomy is done near the origin on the periosteum, and approach is either through the skin or through the conjunctiva. Five prism diopters, with the eye in the primary position, is the least correction obtained from tenotomy of the inferior oblique muscle, according to White. For a smaller effect he opens the muscle sheath, resects 1 cm. of muscle and leaves the sheath. Dunnington gave from 10 to 15 prism diopters as the average correction and 22 prism diopters as the upper limit of correction. He has not observed a serious overcorrection. Rugg-Gunn cuts the inferior oblique muscle from its origin and resutures it to the periosteum ¼ inch (0.6 cm.) posterior to its original attachment. O'Connor favors shortening the superior rectus muscle by means of the cinch shortening operation. After advancing the paretic muscle Peter brought the upper halves of the external and the internal rectus muscle up to the stump of the superior rectus muscle. For detailed discussions of this subject the reader is referred to articles by White and Dunnington.

For paralysis of the inferior rectus muscle most authors (Dunnington, White, Berens) have advised resection of the paretic muscle. When there is secondary spasm of the direct antagonist White advised recession of the superior rectus muscle of the same eye. For secondary deviation of the other eye Berens has suggested retroplacement of the superior oblique muscle. Wiener described cutting off the inferior oblique muscle and sewing it to the sclera, nasal to and in front of the inferior rectus muscle.

The operation of choice for a paralysis of the superior oblique muscle is recession of the inferior rectus muscle of the other eye (Berens, Dunnington, Argañaraz, McMullen, White). Dunnington and Berens use tenotomy of the inferior oblique muscle of the same eye when this muscle is overactive. Savin prefers to do a recession of the superior rectus muscle of the same eye. Vasquez Barrière follows the method of Jackson in transplanting the scleral insertion of the superior rectus muscle of the same eye. J. M. Wheeler has given a method for shortening the superior oblique muscle. The superior rectus muscle is temporarily tenotomized, and the globe is rotated downward. A double-armed suture is placed in the middle third of the tendon of the superior oblique muscle, several millimeters from the insertion. The needles are then secured into the superficial sclera, temporal to the insertion, the tendon being thus folded on itself and advanced. Rules for the amount of advancement cannot be given, but it can be graded, and immediate overcorrection is advised. Aurand treated a patient

with complete paralysis of the superior oblique muscle resulting from a stab wound. He divided the external rectus muscle and the inferior rectus muscle longitudinally and advanced the lower and the outer half, respectively, to a point 3 mm. from the limbus at 7:30 o'clock. There was complete disappearance of the squint and the diplopia. Suker has used the same method, with a good functional result.

For paralysis of the inferior oblique muscle, recession of the superior rectus muscle of the other eye has been advocated by a number of authors (Berens, White). Wheeler has described an advancement of the paretic muscle which has given excellent results. Through an incision in the skin the muscle is isolated in the usual fashion and is cut off from the periosteum. It is then carried over the orbital margin and sutured onto the anterior surface of the malar bone with two mattress sutures. A portion of the muscle may be resected at the same time.

The question of the relation between vertical strabismus and lateral strabismus and their surgical treatment has not been settled. It has been fully discussed by White, who, as a rule, favors correcting the vertical deviation first if it is over 5 prism diopters. Others believe that if the lateral deviation is of considerable size there is little hope of a cure from the removal of the vertical deviation alone and that the lateral and the vertical deviation should be corrected at the same time.

SUMMARY

The literature of the last seven years on the surgical treatment of strabismus has been reviewed. An attempt has been made to evaluate the methods in current use and to give the procedure favored by the majority of ocular surgeons. Some of the newer operations are briefly described.

In conclusion, it can be said that there is no unanimity of opinion as to the proper handling of strabismus. Comparably favorable end-results can be achieved by several methods. The procedure of choice for any surgeon is the one with which he is most familiar and in the performance of which he is most skilful.

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Obituaries

FÉLIX DE LAPERSONNE
1853–1937

Among the ophthalmologists of the past half-century, none was more highly esteemed by his colleagues than the former head of the ophthalmic clinic of the Hôtel-Dieu in Paris.

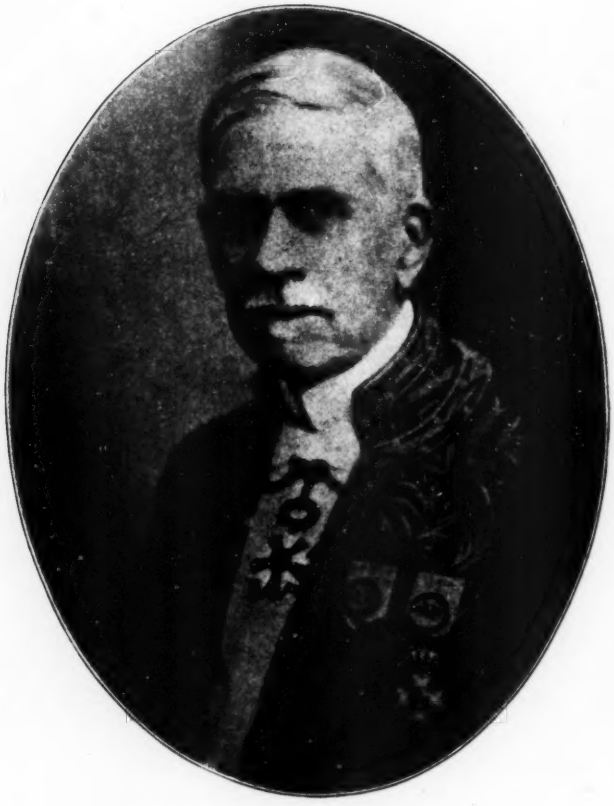
Félix de Lapersonne was born in 1853 in Toulouse, where he took his medical training. Subsequently he became an intern and later chief of staff under Panas in the ophthalmic department of the Hôtel-Dieu. After serving as clinical professor of ophthalmology at Lyons, he succeeded Panas in his clinic at the Hôtel-Dieu. This hospital, one of the most ancient in Europe, dating back to the seventh century, is situated in the heart of old Paris on the Isle de la Cité and facing the Place du Parvis Notre Dame. It has a clinic that is so enormous and of such a varied character that students and clinicians are drawn to it from the most remote French possessions.

I remember with keen pleasure the courtesy and consideration with which de Lapersonne, years ago, demonstrated rare tropical diseases affecting the eye to a small group of foreign visitors. His courtesy laid the foundation of a friendship that continued to the end. Some of his students became in time the heads of other important clinics, and such was their devotion to the master that only a word from him was sufficient cause for them to go to infinite trouble to illustrate unusual methods or rare conditions to those who were his friends.

This great teacher was a critical and discriminating observer. There was no branch of ophthalmology which he had not enriched by his contributions. There is no name occurring more frequently in the ophthalmic bibliographies of the past decades than his.

The simple elegance of his manner and his personal charm made him a distinctive figure in any gathering in which he might be present. He visited the United States once only, when he served as vice president of the International Congress of Ophthalmology, held in Washington, D. C., in 1922, and made one of the most important addresses on that occasion. He received a warm welcome and made many friends, but a trans-Atlantic voyage caused him such acute suffering that he could never be prevailed on to attempt it again. He received numerous distinctions, was president of the French Académie de Médecine and a commander of the Legion of Honor.

At the close of the Thirteenth International Congress of Ophthalmology, held in Netherlands in 1929, the International Association for the Prevention of Blindness was organized, of which he was made president. His judgment and organizing skill were of great value in bringing together those whose friendship had been shaken by the great war. An incident which occurred at that time will illustrate his tact and magnanimity. The names were being read of those who would represent the different countries. After the reading was finished, van



FÉLIX DE LAPERSONNE
1853-1937

der Hoeve, who had called the group together, whispered, "Germany seems to be left out. That will not do." I, feeling much the same way, quietly called de Lapersonne's attention to the omission. "Wait," said he, "wait!"

Then, other business having been transacted, de Lapersonne proceeded to say: "The acceptance by a representative from Germay for a position on the council came too late to be included in the list as read. I, therefore, as president of the association, take pleasure in presenting

the name of Professor Axenfeld to represent Germany." The proposal was approved by acclamation, and as de Lapersonne, reaching across the table, grasped the hand of Axenfeld, a thrill went through the group. The animosities which the war had engendered were forgotten. The breach between these two great ophthalmologists was closed.

After his retirement from active work nothing more deeply appealed to de Lapersonne than the widening of the field for the prevention of blindness, a work which his prestige carried throughout the world. When, therefore, the Leslie Dana Gold Medal was presented to him, it gave him great pleasure—not, of course, that any distinction could add to those which his ability and fame had already brought to him, but he was especially gratified to have come to him this evidence of the high regard in which he was held by his friends across the sea. As the medal was pinned on his breast, with the beautiful gesture so spontaneous to the French, he leaned forward and kissed me on each cheek.

In the passing of Landolt, of Abadie, of Morax, and now of de Lapersonne, all who have gone during the past decade, France has lost some of her great teachers, but it is gratifying to know that their pupils, the younger French workers, with newer methods will probably solve many of the problems to which ophthalmologists as yet do not know the answers.

PARK LEWIS.

DR. MED. SØREN HOLTH
1863-1937

Not only ophthalmology in northern Europe but the whole ophthalmologic world has suffered a great loss in the death of Søren Holth on September 23.

Holth was born on July 21, 1863, and died at the age of 74 years, the last of the three great Norwegian ophthalmologists—Johan Hjort, Hjalmar Schiötz and Søren Holth.

SØREN HOLTH, M.D.
1863-1937

Holth was graduated from medical school in December 1891 and received his M.D. degree in December 1896, after having practiced as an ophthalmologist in Drammen for about four years. His thesis, "The Indirect Fixation Blindness of the Eye," was based on the material he collected in Drammen. He settled in Oslo (then called Kristiania),

where he practiced until two years ago, when increasing otosclerosis forced him to retire.

For six years he was first assistant at the university ophthalmologic clinic and acted as locum tenens for Professor Schiötz when the latter was absent. When Schiötz resigned, the chair was offered to Holth, but because of his difficulty in hearing, Holth did not accept the position. Through repeated visits to foreign clinics he continued his studies—even in recent years. He frequently received invitations to demonstrate at various clinics his methods of operating or to read papers, notably at Oxford, Helsingfors, Stockholm and Budapest. The last named city he visited as late as 1932.

His power for work was phenomenal, as witnessed by his one hundred and ten scientific publications. His chief interest was in the study of glaucoma. In his iridencleisis antiglaucomatosa he has left a lasting monument.

Holth was in every respect a courageous man; he never was afraid to try new methods invented and first used by others. He never hesitated to give an opponent the honor he deserved if he found him in the right and himself in the wrong. As a *curiosum* of his immense operative practice it may be mentioned that he, as far as is known, was the only ophthalmologic surgeon to perform a reclinatio cataractae on a lioness—and with good results!

Holth had many interests besides his medical studies. He also had many friends, but he declined to participate in politics. The old poet's words *bene vixit qui bene latuit* are fitting here. A sincere friend has passed, and those who had the privilege of his friendship have suffered a great loss.

HARALD G. A. GJESSING.

Correspondence

TREATMENT OF NEUROPARALYTIC KERATITIS BY CLOSING THE LACRIMAL CANALICULI

To the Editor:—In the September issue of the ARCHIVES, Drs. J. A. MacMillan and W. Cone (Prevention and Treatment of Keratitis Neuroparalytica by Closure of the Lacrimal Canaliculi: Report of a Case, ARCH. OPHTH. **18:** 352 [Sept.] 1937) reported a case in which neuroparalytic keratitis following section of the posterior root of the fifth nerve for neuralgia was relieved by cauterization of the lacrimal puncta. Commenting on this result, they stated, "It is comparable to the results described by Beetham after he had closed the ducts in patients with filamentary keratitis." It seems to me that this statement gives too little credit to Beetham, who was the first to employ this procedure to relieve dryness of the eye due to insufficiency of lacrimal secretion. The writers did not state whether or not it was Beetham's communication that led them to employ the procedure in their case.

The writers contended that if after operation for trigeminal neuralgia the tearing is greatly diminished and the conjunctiva congested, the canaliculi should be closed (presumably by cauterization). Since in many cases of this condition the secretion of tears is restored, sometimes at an early date and sometimes only after the lapse of a year, it would seem that such a radical procedure should be postponed as long as possible. Simply tying off the canaliculi would seem to be a better procedure at the outset, and this could be repeated when necessary. When a permanent effect is desired, I have found in cases of filamentary keratitis that extirpation of the canaliculi is the best procedure. After inserting an ordinary pin in the canaliculus, the canaliculus can easily be dissected out, with the sacrifice of little, if any, additional tissue.

The writers stated, "Our studies have convinced us that although the cornea is anesthetic the diminished secretion of tears is the primary factor in the evolution of these corneal lesions." The only evidence, however, that they gave on which they could base this conviction is the satisfactory results obtained in their case and in the case reported by me in which I kept the cornea continually moistened with Ringer's solution. They failed to mention that in the same communication (Verhoeff, F. H.: The Cause of Keratitis After Gasserian Ganglion Operation, *Am. J. Ophth.* **8:** 273, 1925) I stated that in six cases of keratitis following operation on the gasserian ganglion I was unable to detect any lacrimal secretion. If they had not ignored this statement, perhaps their conviction would have been still stronger.

The writers described a new method for measuring the rate of secretion of tears, which seems adequate for the purpose desired, but they brought forward no evidence that this method is superior to others. They stated that they have employed this method in the case of "every patient who has been operated on for trigeminal neuralgia in the Montreal Neurological Institute in the past year." Most astonishing is the fact that the only statements they made as to the results they obtained

are that these have varied in the same patient, and that they obtained "definite information as to the presence of secretion and a fairly accurate impression of its amount." They did not state that they employed it in the case which they reported or in any case in which there was keratitis. In other words, except for the evidence furnished by the fact that the keratitis was relieved by cauterization of the puncta in the single case they reported, they offered no new evidence that this type of keratitis is due to dryness.

The writers stated that the findings in their case contradict my theory that the cause of the dryness in cases of the condition in question was due to injury to the large superficial petrosal nerve, since, so they asserted, this nerve was not injured in their case. They gave no evidence, however, that this nerve was not injured, and therefore the evidence that I brought forward as to injury to this nerve in such cases remains as strong as if their case had not been reported. This evidence is so strong that, in my opinion, it proves conclusively that the large superficial petrosal nerve was injured in their case. Presumably their statement to the contrary is based on a supposition of the operator, but such a supposition is without importance in view of the fact that most often, even when the facial nerve is injured during an operation on the gasserian ganglion or one of its roots, the operator is not aware of the fact at the time.

F. H. Verhoeff, M.D., Boston.

News and Notes

EDITED BY DR. JOHN HERBERT WAITE

GENERAL NEWS

Annual Course, Research Study Club of Los Angeles.—The Research Study Club of Los Angeles will hold its regular midwinter course in Ophthalmology and Otolaryngology from January 17 to 28, inclusive. The fee, as usual, will be $50. The teaching staff will include Prof. Alfred Bielschowsky, Dr. Edward Jackson, Dr. John O. McReynolds and Mr. Frederick Jobe, on the eye, and Dr. Arthur W. Proetz, Dr. Grant Selfridge, Dr. W. P. Covell, Prof. Vern O. Knudsen, Dr. Simon Jesberg and Dr. Louis K. Guggenheim, on the ear, nose and throat. In addition, there will be twelve instruction courses. The first week will be devoted mostly to the eye, and the second mostly to the ear, nose and throat.

SOCIETY NEWS

Philadelphia County Medical Society, Eye Section.—At the scientific session of the Eye Section of the Philadelphia County Medical Society held on Nov. 4, 1937, at the Wills Hospital, the clinical meeting was in charge of Dr. Alvin W. Howland, chief resident physician. The chief and his assistants presented a contemporaneous group of demonstrations on external disease, pathologic conditions of the fundus, surgical results, research apparatus and clinical facilities, from which members and guests selected their preference.

CORRECTION

In the article by Dr. Avery DeH. Prangen entitled "Some Problems and Procedures in Refraction" (ARCH. OPHTH. **18**: 432 [Sept.] 1937, an error occurs in the third sentence on page 441. This sentence should read: "One can easily measure the residual accommodative power at the conclusion of a refraction in which cycloplegia was employed by adding a + 3.00 D. lens to the findings and measuring the accommodative range, the difference between the far point and the near point, each expressed in diopters."

The fourth sentence on this page should be omitted.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Biochemistry

DISTRIBUTION OF SODIUM CHLORIDE AND DEXTROSE IN PLASMA AND AQUEOUS. Y. DERRIN, G. JAYLE and P. FRIZT, Compt. rend. Soc. de biol. **125:** 148, 1937.

The authors hold that the theory of dialysis as the source of the formation of aqueous, as suggested by Mestrezat and elaborated by Duke-Elder, requires modification, since the dextrose content of the aqueous is consistently lower than that of the plasma.

J. E. LEBENSOHN.

THE AMYLASE OF THE SUBRETINAL FLUID. H. J. M. WEVE and F. P. FISCHER, Arch. f. Augenh. **110:** 390, 1937.

Having determined that a sugar-forming ferment, which rapidly disappears, often occurs in the subretinal fluid found in cases of retinal detachment, Weve and Fischer have shown that this ferment is a desmoamylase, which appears to originate in the disturbed retina. The quantity of fluid is small and presents obvious difficulties in chemical analysis. The authors describe a new method of micro-analysis for the examination of subretinal fluid. They conclude that the amylase of the subretinal fluid is a peroxydamylase. Its action in the synthesis of sugar is inhibited by cevitamic acid. They believe that measurements of the catalase index of the blood is evidence that the substance is not present in the blood.

E. G. SMITH.

Congenital Anomalies

FAMILIAL MACULAR COLOBOMATA. P. JAMESON EVANS, Brit. J. Ophth. **21:** 503 (Sept.) 1937.

The author found macular colobomas in five members of a family. In this family no defects of the limbs and no other physical defects were found, and apart from the ocular conditions all these persons were normal.

The next generation so far has shown no similar defects. Unfortunately, the previous generation cannot be fully investigated, but the suggestion is that the defect in this family derived from the maternal side. Further observations in the side branches on this side of the family may possibly disclose instances of the same defect.

W. ZENTMAYER.

HEREDITARY DEVELOPMENTAL ERRORS AS CAUSE OF LOWERED RESISTANCE. W. LÖHLEIN, Arch. f. Ophth. **136:** 434 (Feb.) 1937.

The assumption that eyes with hereditary developmental errors are predisposed and less resistant to inflammatory or degenerative diseases acquired intra vitam is illustrated with three instructive but hardly conclusive reports of cases.

P. C. KRONFELD.

Cornea and Sclera

A HITHERTO UNDESCRIBED BILATERAL, SYMMETRICAL, SUBEPITHELIAL CORNEAL OPACITY IN A CASE OF PAGET'S OSTEITIS DEFORMANS: REPORT OF A CASE. F. PEPPMÜLLER, Klin. Monatsbl. f. Augenh. **98:** 1 (Jan.) 1937.

A man aged 62 had suffered from progressive fibrous osteitis deformans, Paget's osteitis, since 1918. All the bones of the skeleton became involved, including those of the skull, as is evident from pictures. Peppmüller observed the eyes since 1929. Two symmetrical subepithelial opacities in Bowman's membrane were seen in each eye, one near the temporal, the other near the nasal, limbus. These opacities, which were oval and brownish were composed of dotted and linear spots. Fine deposits of melanin were seen in the conjunctiva outside the limbus, adjoining the areas of the corneal opacities but not connected with them. They were progressive from the start, while the corneal opacities remained more or less stationary until 1933. Since that time they had progressed, finally becoming confluent in the center of the cornea of each eye. The opacities did not consist of melanin but may represent a degenerative procedure. It could not be decided whether they were caused by arteriosclerotic changes or by the specific metabolic changes of Paget's disease.

K. L. STOLL.

Experimental Pathology

EXPERIMENTAL REPRODUCTION OF OCULAR LEPROSY. A. SANTONASTASO, Ann. di ottal. e clin. ocul. **65:** 321 (May) 1937.

Review of the literature revealed uncertainty as to whether leprosy can be transmitted to the eyes of animals. Santonastaso inoculated ten rabbits' eyes with fragments of material from active lesions of the conjunctiva and cornea of patients with leprosy. Inoculation was made into the anterior chamber. The eyes were removed at the death of the animal or after varying periods. Aside from the usual histologic stains, a careful search for bacteria was made. In most cases a certain amount of inflammation involving the iris and cornea followed the inoculation, but the signs of active inflammation usually subsided after several weeks. In one case the implanted tissue continued to increase in size, and histologic sections showed that vascular connections had been established between it and the neighboring iris. In this specimen alone large numbers of leprosy bacilli were observed. In the other cases the inflammatory changes observed were considered to be nonspecific and probably due to absorption of the foreign material implanted in the

chamber. They were accompanied by degenerative changes in the iris and organization of inflammatory products. The stain for bacteria in these cases showed either absence of organisms or a relatively small number of degenerated bacterial forms.

The author concludes that the possibility of inoculating rabbits with leprosy was not definitely determined in his work and that such inoculation if possible occurs only under special conditions. A bibliography is given.

S. R. GIFFORD.

Glaucoma

THE SURGICAL TREATMENT OF CHRONIC GLAUCOMATOUS OCULAR HYPERTENSION. HENRI LAGRANGE, Brit. J. Ophth. **21:** 477 (Sept.) 1937.

This article is written in commemoration of the thirty-third anniversary of subconjunctival and limbic sclerotomy. There is a general consideration of the symptoms, pathogenesis and treatment of chronic glaucomatous ocular hypertension. In connection with the pathogenesis Lagrange says:

"The tension retaining the aqueous humor within the eyeball seems to be specially governed by the action of the *pars ciliaris retinae*. In 1883, Boucheron presented to the French Ophthalmological Society an initial contribution to this point, contemporary with the work of Ranvier maintaining that the epithelial elements were superficial glands. These findings are confirmed by pathological histology. The presence in the ciliary body of adenomas, epitheliomas and carcinomas, described by F. Lagrange in his treatise on tumours of the eye, tends to affirm the existence of a differentiated tissue whose structure shows that it is actively concerned in elaborating the aqueous humor.

"The sympathetic is the nerve of secretion, it increases ocular tension when stimulated and the decisive experiment by Pourfour du Petit and Claude Bernard supports the entire physiology of the sympathetic in its relations with the eye. The individual designation of the secretory nerves is due to the work by Dastre and Morat in 1878 and studies showing their autonomy and functional individuality were made by Heidenhain. It evidently remains to define the nature of the profound causes which may precipitate hypersympathicotony, but the foregoing suffices at least to render homage to the theory of 'secretory neurosis' suggested by Donders with reference to glaucoma.

"The alteration in the evacuation of the ocular liquids from the corneoscleral shell becomes added more or less rapidly to the condition of hypersecretion often occurring during the first stages of the disease.

"Secondary trophic disturbances affecting the vessels of the retina and involving the optic nerve add new complications, in varying degrees. These complications result from a vitiated hydrostatic condition and are beyond the control of the mechanism regulating the intra-ocular circulation. All causes which favour vascular sclerosis, especially syphilis and senility, constitute aggravating factors in the progress of these trophic disturbances secondary to hypertension occurring in a closed chamber, which finally produce optic atrophy and the papillary excavation of Schnabel.

"In brief, though from the viewpoint of general pathology there still remain in the pathogeny of glaucoma certain obscure points referring to the true causes of the sympatheticotonic disturbances, the metabolic changes and the liquid exchanges, one fact is shown by clinical observation, namely, that glaucoma is related to nervous, organic, psychic and doubtless humoral instability, and to a so-called diathesis. It has the characters of an 'essential disease,' whose effects vary according to defective functioning which is individual, congenital or acquired with advancing age, the variations constituting factors which have great influence upon the course of the disease. Of its factors, standing in the order of their importance, hypertension occupies the first place and the therapeutic problem is *to prevent vitiation of the liquid exchanges by medical treatment or to avert, by surgical measures, the retention of liquids which is fatal for the neuro-retinal structures.*"

In acute glaucoma the value of iridectomy is unquestionable. Von Graefe has placed in the hands of ophthalmologists a curative procedure which renders his name immortal. The technic of the modified operation of Felix Lagrange as practiced by the author since 1926 is described and illustrated. From results for patients followed for at least one year the conclusion is sincerely warranted that the fistulizing method succeeds in 85 per cent of cases of chronic glaucoma, while iridectomy is successful in an average of from 25 to 30 per cent of cases, according to the best statistics.

W. ZENTMAYER.

CYCLODIALYSIS. L. HEINE, Klin. Monatsbl. f. Augenh. **97**: 721 (Dec.) 1936.

Heine refers to two contributions on cyclodialysis which appeared in 1935, both of which demonstrated progress. The first, that of E. von Grósz, showed that cyclodialysis has been more and more practiced at the University Eye Clinic at Budapest during recent years. Cyclodialysis is taking increasingly the place of trephining. The second publication is that by Vannas, whose experiences with this operation were very satisfactory. The opinions and results of other operators, such as Elschnig, Salus and E. Fuchs, are quoted by Heine. He points out that it is possible to operate more exactly with the technic of cyclodialysis than with the trephine operation and emphasizes the superiority of the former over the latter.

K. L. STOLL.

Hygiene, Sociology, Education and History

PROTECTIVE GOGGLES FOR PHYSICIANS. M. H. VIALLEFONT, Arch. Soc. d. sc. méd. et biol. de Montpellier **18**: 25, 1937.

Protective glasses for physicians are more needed than is generally realized, in proof of which various cases are cited. In curetting an infected uterus a gynecologist received septic particles in the eye, and severe inflammation resulted. A laryngologist opened a retropharyngeal abscess with a like consequence and loss of considerable visual acuity

as a final result. A pediatrician opening an ampule of vaccinia virus had some droplets fly in his eye; vaccinia followed, necessitating enucleation of the eye later.

J. E. LEBENSOHN.

MORE UNPUBLISHED LETTERS OF OPHTHALMOLOGISTS ADDRESSED TO HORNER DURING 1856-1885. A. BADER, Klin. Monatsbl. f. Augenh. **97**: 787 (Dec.) 1936.

After a short introduction Bader publishes twenty-four letters addressed to Horner from eminent ophthalmologists, such as Troeltsch, A. von Graefe, T. Leber, Michel, Donders, Schweigger, Schmidt-Rimpler, Zehender, Wilhelm Hess, Laqueur, Herman Knapp, Landolt, A. von Hippel and others. Interesting side-lights of a professional and personal nature may be gathered by the reading of these letters. It may be mentioned that Leber described the condition at A. von Graefe's ophthalmic clinic after the death of this eminent man and that H. Knapp referred to extraction of cataract with and without antiseptics in 1881, the year which brought him his hundred thousandth patient.

K. L. STOLL.

Lacrimal Apparatus

DACRYOCYSTORHINOSTOMY. R. E. WRIGHT, Lancet **2**: 250 (July 31) 1937.

As the otherwise excellent method of Dupuy-Dutemps proved too lengthy for the busy clinic in India, Wright sought for a quicker and easier method. The new method is characterized by a small facial incision, and the steps are shown in diagrams with explanatory notes. The difficulty in this operation, and particularly in this method, is the suturing of the cut edges in the sac and in the nasal mucosa. Any fine, long needle holder and a large-eyed, fully curved needle measuring from 6 to 7 mm. from the point to the eye will serve. The procedure is made easier by the adoption of lateral hinged flaps. A straight, eyeless needle from 3 to 5 mm. long (with suture material attached) is easiest to pass a point of suture in a small deep hole. The bone in the floor of the lacrimal groove of the sac is removed, and the hinge flaps are approximated and united by two points of suture. The anterior lip of the opening of the sac is then sutured to the periosteum of the crista. The cutaneous wound is sutured.

Wright says that this method is as bloodless as the ordinary operation and is preferable to extirpation in conditions suited for dacryocystorhinostomy, e. g., a dilated sac in a middle-aged patient. A study of the diagrams will explain the method.

ARNOLD KNAPP.

LONGITUDINAL AND TRANSVERSE SUTURE OF THE LACRIMAL CANALICULI: REPORT OF A CASE. K. ASCHER, Klin. Monatsbl. f. Augenh. **97**: 770 (Dec.) 1936.

A boy aged 6 years fell against a door-knob and suffered a triangular wound in the upper eyelid. The lid was partly bruised, and the upper canaliculus was severed near the tear point. Ascher closed the wound

with a transverse suture according to the method of Elschnig, after the insertion of a thin probe. The technic is described in detail. The canaliculus was permeable after six days and remained open. Primary suturing of the severed canaliculus is essential, in the author's opinion. Only a transverse suture seems to yield good results, as suggested by Wagenmann in 1913. It appears as if no case in which a successful transverse suture of the *upper* canaliculus was made has hitherto been published.

K. L. Stoll.

Lens

Striations of the Aging Nucleus of the Lens. H. Goldmann, Arch. f. Augenh. **110:** 405, 1937.

Goldmann has studied the striations of the aging nucleus of the lens and found that the relations which exist in the anterior part are qualitatively similar to those in the posterior part. Exact measurements were not made. The so-called superficial surface of the aging nucleus is designated by the author as a reflex zone layer of varying thickness, which in many cases may be resolved into its component elementary layers. Goldmann believes that the nucleus increases in size with age and its nuclear or inner border appears to move inward, while the outer border moves toward the capsule. This is apparently the result of repeated superimpositions of recurring bands of disjunction. In the aging lens the reflex zone may be divided into three zones, corresponding to three separate periods of life. These are important in the determination of the time at which the processes occurred in the pathologically changed lens.

E. G. Smith.

Intracapsular Extraction of Cataract in Diabetic Patients: Report of Cases. E. Purtscher and H. Dibold, Klin. Monatsbl. f. Augenh. **98:** 24 (Jan.) 1937.

Extensive studies on intracapsular extraction of cataract in diabetic patients were made jointly by Purtscher, of the ophthalmic division, and Dibold, of the division for metabolic diseases, at the Hospital of the City of Vienna. The authors report in detail on thirty-seven intracapsular extractions of cataract performed on twenty-nine diabetic patients from 1933 to 1936. The data are recorded on tables which refer to the age of the patient, the type of cataract, the condition of the fundus and of the retinal vessels, the complications and results of the operation and the blood pressure and which state whether insulin was given or not. The following complications met with during the operation received special consideration: severe hemorrhages from the conjunctival vessels, dark coloration of the aqueous by pigment of the iris, postoperative rupture of the wound and postoperative irritation of the iris. Simultaneous observation of the metabolic and ophthalmologic conditions yielded the following conclusions:

1. Hemorrhages are apt to occur during the operation, especially during the incision and the formation of flaps, in persons who had to undergo preparatory treatment with insulin, after rapid reduction of

the blood sugar content prior to the operation and in the presence of hemorrhagic retinitis with or without hypertension (increased blood pressure).

2. Accumulation of pigment of the iris in the anterior chamber may be expected in chronic diabetes.

3. Irritation of the iris is imminent in the presence of a high content of blood sugar, in faulty compensation of the diabetes and simultaneous hypertension (high blood pressure).

4. A comparison of diabetic patients with cataract with nondiabetic persons with cataract shows that a much larger percentage of the former have a high degree of hypertension.

5. The proneness of diabetic patients to hemorrhage during the operation is not dependent on the blood pressure.

6. Flooding out of pigment and postoperative irritation of the iris are more frequent in diabetic patients than in patients with normal metabolism.

7. No difference in the result of the operation and in recovery was found between diabetic patients with cataract and healthy patients with cataract after proper preoperative care, and the visual results were practically the same.

K. L. STOLL.

Lids

CHALAZION: ITS TRUE NATURE AND ITS RELATION TO AMETROPIA. G. ROBERT, Ann. d'ocul. **174:** 473 (July) 1937.

In a recent article on this subject Solignac mentioned that in 1933 Robert had drawn attention to the influence of ametropia on the development of chalazion. Since the publication of his article the author has had time to verify statements made then and has arrived at the following conclusions:

The chalazion is a retention cyst, appearing always in eyes which show even slight ametropia. Even when large, it will disappear in nearly all cases after a varying period if one corrects the metropia. The chalazion should be cured by optical and medical treatment. It is not an organic tumor. The deformities and modifications of the secreting cells are not surprising when one considers the phenomenon of retention.

S. H. MCKEE.

Neurology

SYPHILITIC ARACHNOIDITIS OF THE OPTIC CHIASM. L. HAUSMAN, Arch. Neurol. & Psychiat. **37:** 929 (April) 1937.

Hausman has made a careful study of syphilitic chiasmal arachnoiditis per se and in relation to tumors of the brain and other chiasmal lesions. Five cases of syphilitic chiasmal arachnoiditis and seventy-six cases of verified tumor of the brain supplied the data for the analysis. Because of the infrequent reports of this condition in the ophthalmic literature, as contrasted with the frequent reports of nonsyphilitic arachnoiditis, the article should be of interest to every ophthalmologist. As the

author's conclusions so adequately summarize the paper, stressing important points not generally considered, they are quoted directly:

"1. The syndrome of syphilitic chiasmal arachnoiditis may manifest itself clinically in one of two ways: (1) as the classic form of primary atrophy of the optic nerve, with heteronymous visual field defects, which occurred in cases 1, 2, 3 and 4, or (2) as the less frequent form of papilledema without internal hydrocephalus or increased intracranial pressure, which occurred in case 5.

"2. The visual fields in cases of syphilitic arachnoiditis may show hemianopic defects or concentric constriction or both. Blindness may also occur in one or both eyes. The visual impairment resembles that associated with chronic nonsyphilitic cisternal arachnoiditis and expanding lesions of the chiasmal region and when it is associated with serologic evidence of syphilis, even if this is of the blood alone, there is a strong possibility that the underlying condition is syphilitic arachnoiditis of the chiasm. For that reason the interpretation of the hemianopic visual field defects reported in the literature in questionable cases of tabes associated with atrophy of the optic nerve is open to question. The inclination in some quarters to regard the presence of primary atrophy of the optic nerve alone in cases of syphilis, without other neurologic signs, as evidence of tabes is contrary to all sound neurologic teaching. Many of these cases may belong to the group of instances of syphilitic chiasmal arachnoiditis, which would account for the heteronymous peripheral field defects reported in cases of this condition.

"3. Given the classic chiasmal syndrome of primary atrophy of the optic nerve with heteronymous visual field defects, the problem which arises is one not of localization (the chiasmal site of the lesion is evident) but of differential diagnosis, for the etiologic factor may be one of many: (*a*) an expanding lesion (tumor, cyst or aneurysm), which may be intrasellar, suprasellar or parasellar; (*b*) arachnoiditis (syphilitic or nonsyphilitic); (*c*) trauma or (*d*) heredodegeneration. Syphilis as an etiologic factor should be carefully considered in each case. There were twelve instances of the chiasmal syndrome in the series of cases of combined syphilis and tumor. In three cases positive serologic reactions were shown, and in the remaining seven the condition proved to be a neoplasm in or near the sella.

"The problem of etiologic differentiation may become complicated when certain factors of neoplasm and syphilis exist together in the same case. If a positive Wassermann reaction, either of the blood or of the spinal fluid, is present with the clinical setting of neoplasm of the brain, the likelihood that a tumor other than gumma is associated with syphilis is exceedingly slight. When the factor of syphilis has been eliminated in cases of the chiasmal syndrome of primary atrophy of the optic nerve and visual field defects, the problem of differential diagnosis becomes more difficult, for it is not always possible to differentiate between nonsyphilitic chronic cisternal arachnoiditis and suprasellar tumor with normal roentgenographic findings in the skull. In many such instances of nonsyphilitic primary atrophy of the optic nerve in which exploration has been made for a possible hypophysial neoplasm, the condition has proved to be chronic cisternal arachnoiditis. The relation of hereditary atrophy of the optic nerve (Leber's disease) to chiasmal arachnoiditis is also discussed.

"4. The nature of the pathologic process in syphilitic arachnoiditis is such that it compresses the optic nerves and chiasm. Why this local process around the chiasm should produce atrophy of the optic nerve in one instance and papilledema in another remains obscure. Reference is made to the dilatation of the chiasmal cistern described by others in cases of chronic cisternal arachnoiditis.

"5. The chief problem which confronts both the patient and the physician is the prevention of blindness which may develop within two years. The choice lies between vigorous antisyphilitic medication and surgical intervention to free the adhesions around the chiasm. The former gives little promise of relief; surgical treatment appears to be the method of choice, particularly when blindness is imminent."

R. IRVINE.

PRIMARY DEMYELINATING PROCESSES OF THE CENTRAL NERVOUS SYSTEM. A. FERRARO, Arch. Neurol. & Psychiat. **37:** 1100 (May) 1937.

This is a special review article summarizing the conditions mentioned in the title. The demyelinization which constitutes the fundamental pathologic changes of diffuse sclerosis has no particular location, extending at times from the frontal to the occipital pole in more or less symmetrical fashion. As a result the entire nervous system is affected, and the patient becomes practically a decerebrate animal.

It is noteworthy that, with other symptoms, in adults initial symptoms generally consist of disorders of vision and acute blindness and occasionally hemianopia, paresthesias, severe ataxia, and sensory disturbances of objective and subjective types. In the fully developed condition acute blindness may appear, associated with optic neuritis and choked disks and partial or total atrophy of the optic nerve. In some cases the fundi are normal. To and fro swinging movements of the eyes have been reported, and external ophthalmoplegia due to involvement of the third nerve is frequent. Involvement of the sixth nerve has been reported. Nystagmus is not common. Paresis of conjugate lateral gaze and hemispasm of the eyelids have been described.

In children disorder of vision as an initial symptom has been noted in 12 per cent of the cases and a combination of disorder of vision and of hearing in 6 per cent.

R. IRVINE.

ARACHNOIDITIS OF THE OPTIC CHIASM FOLLOWING CONTUSION, WITHOUT INJURY TO THE RIGHT PALPEBRAL REGION. M. KALT, PUECH and KREBS, Bull. Soc. d'opht. de Paris, May 1937, p. 291.

The authors report an interesting case of arachnoiditis of the chiasmal region with unusual findings. After trauma to the right palpebro-orbital region visual difficulties occurred. In addition to central scotomas there existed double hemianopic bitemporal and paracentral scotomas. Intervention seven months after the accident permitted freeing of the optic nerves, which was followed by partial regression of the scotomas and recuperation of visual acuity. Aside from ecchymosis of the skin at the site of trauma, the patient complained only of photopsia at the time of the accident. One month later the visual acuity of

the right eye was 0.2. There was some recovery of vision, but ten weeks after the accident the visual acuity of the right eye was 0.4, while the left eye remained normal. Drawings of the visual fields made during the course of the condition accompany the article, and campimetric fields of the central areas are shown. Operation revealed active arachnoiditis, and vision returned immediately. The authors express the opinion that the lesion was due to belated infection of the meninges following trauma.

L. L. MAYER.

Ocular Muscles

THE PRESCRIBING OF PRISMS IN EVERYDAY PRACTICE. ANDRÉ HUDELO, Ann. d'ocul. **174:** 528 (Aug.) 1937.

Ophthalmologists of different countries have studied from time to time the problem of heterophoria, that is, the subjective disturbance in the convergence-divergence function. In the last few years many authors, English, American and German, and particularly Landolt, in France, have studied the question from both a theoretical and a practical angle.

Hudelo takes up: (1) what class of patients will benefit from the clinical examination for heterophoria; (2) the determination of the state of the convergence-divergence function, with a consideration of the principles, technic and results; (3) the clinical deductions, and (4) the results of examination.

Among seventy-three subjects with functional disorder of vision studied, thirty-two were troubled with only uncertain disturbance of the convergence-divergence function, for which no prisms were prescribed; eight were cured by prisms; eight have not been seen again, and four have had no relief. Seventeen have not appeared to benefit from prisms, for various reasons. The treatment in cases of emmetropia, those of slight ametropia and those of more pronounced ametropia is then discussed, and replies are given to certain possible objections.

S. H. MCKEE.

Operations

THROUGH AND THROUGH OPTIC KERATOPLASTY WITH ROTATION OF THE SCAR. R. FRIEDE, Arch. f. Augenh. **110:** 426, 1937.

Friede reports eight cases in which through and through keratoplasty with rotation of the scar was done and evaluates the results in these cases. He discusses the operation and the indications for its use, as well as conditions which must be present in the cornea. The preparation of the patient, the sequela and the complications are delineated. Modifications of the operation are briefly mentioned. The author believes that the operation has much to offer.

E. G. SMITH.

MELIORATION OF LEUKOMA AND REPEATED TRANSPLANTATIONS OF THE CORNEA. V. P. FILATOV, Vestnik oftal. **10:** 637, 1937.

This is a general discussion of melioration of leukoma. Clinical data will be reported later by Filatov's assistants. Filatov believes that

the corneal transplant is a strong physiologic irritant and causes biochemical reconstruction of the opaque cornea by which the corneal elements regain the property of transparency.

Melioration of leukoma, or substitution of part of the leukoma by transparent corneal elements, was first tried by Filatov in 1933. Since the posterior layers of the cornea often remain transparent, he suggests shaving off the anterior layers of the leukoma and substituting for them the transparent anterior corneal layers of the host's cornea. The purpose of the melioration is not to obtain immediate optical results but to create a favorable nutritive field for the corneal transplant which is ingrafted later in penetrating transplantation. If the corneal transplant becomes opaque, another transplant can be substituted, placed near the previous one, so that finally one of the transplants may remain transparent.

Two types of operations for penetrating melioration are described briefly, and three operations for melioration of leukoma by the transplantation of layers are described in detail. Most frequently Filatov uses for this operation the Filatov-Martzinkowsky (FM-2) knife.

O. SITCHEVSKA.

Orbit, Eyeball and Accessory Sinuses

FIVE UNUSUAL CASES OF PROPTOSIS. R. FOSTER MOORE, Brit. J. Ophth. **21**: 465 (Sept.) 1937.

The author points out the difficulties in the way of determining the causes of proptosis, especially when the condition is unilateral. Palpation is difficult, and surgical exploration necessitates trauma to muscles and nerves, while removal of the external wall of the orbit is an operation of some magnitude. Roentgenograms are of great value, and their findings may prove conclusive in the way of furnishing evidence of diseases of the sinuses, involvement of bone, pressure due to oxycephaly, or enlargement of the optic canal. They may, however, reveal no abnormality when, as is often the case, soft tissues alone are involved. The pulsation of a prominent eye greatly limits the possible causes. The past history, perhaps even a remote one of removal of a breast or a growth elsewhere, may give the necessary hint that one is dealing with a metastatic deposit in the orbit; the serious illness of the patient, with signs of thrombosis of the cavernous sinus, may make the cause clear, or the site and severity of an injury may make it certain that hemorrhage into the orbit has occurred. There is a group of conditions which are insufficiently recognized because of their rather indeterminate nature.

The suggested causes in the five cases reported are:

In the first, lodgment in the internal carotid artery of an embolus from the auricular appendix or of a plaque from an atheromatous patch in the aorta.

In the second, a misleading red herring drawn across the track of a long-standing arteriovenous aneurysm.

In the third, spontaneous leakage from an atheromatous internal carotid artery

In the fourth, noninfective thrombosis of the cavernous sinus.

In the fifth, necrosis of the internal frontal crest.

The cases are reported in some detail, and comments are made concerning the features leading to the diagnosis.

W. ZENTMAYER.

SYMMETRICAL SYPHILOMA OF THE ORBIT: REPORT OF A CASE. C. PASCHEFF, Klin. Monatsbl. f. Augenh. **97:** 751 (Dec.) 1936.

The case of a woman aged 43 is reported in whom syphiloma (gumma) developed to such a size in each orbit as to produce exophthalmos, and blindness was present in one eye, resulting from atrophy of the optic nerve. The appearance was that of symmetrical pseudotumors. A piece of the tumor in each orbit was excised; these specimens showed fibrous structure and proliferation of connective tissue, with some hyaline degeneration; the blood vessels were thickened and partly obliterated. Spirochetes were not observed in the tumors.

Antisyphilitic treatment brought prompt recovery, but one optic nerve remained atrophic. In pointing out the symmetrical occurrence of these syphilomas Pascheff referred to symmetrical tuberculomas, which he described some years ago. In contrast to the latter, the syphilomas of this patient had connections with the periosteum of the orbit.

K. L. STOLL.

Physiology

CHANGES OF THE BLOOD PRESSURE IN THE CENTRAL RETINAL ARTERY FOLLOWING CHANGES IN THE POSITION OF HEALTHY PERSONS: REPORT OF CASES. A. KAMOGAWA, Klin. Monatsbl. f. Augenh. **98:** 54 (Jan.) 1937.

Kamogawa reports the results of the continuation of his research on the blood pressure in the central retinal artery. The differences in a sitting and in a reclining position were discussed in the previous article (*Klin. Monatsbl. f. Augenh.* **97:** 611 [Nov.] 1936).

In the introduction of the present paper the author analyzes the various factors which bring about hypertension. He then gives a résumé of the recent literature and of the clinical research in this field, including a description of the apparatus, the material and the methods of examination. Several tables and twelve charts are added in illustration. After comparing his results with those of other authors, Kamogawa states the following conclusions:

The changes in the blood pressure in the central retinal artery were measured in ten healthy persons for one hour after the transition from a sitting to a horizontal position. Most of the measurements were taken every ten minutes under simultaneous observation of the variations of the general blood pressure, with the exception of one experimental case, in which the measurements were taken every three minutes. Measurements of the intra-ocular pressure were made simultaneously with those of the blood pressure.

He found:

1. The blood pressure in the central retinal artery increased immediately after change from a sitting to a reclining position, but it decreased gradually in systole and diastole during continuous lying down; this decrease lasted thirty minutes.

2. The first curve of the decrease in the blood pressure began ten minutes after lying down and was steep; the second curve showed a slower declination.

3. The average diastolic blood pressure and the average systolic blood pressure were 7.7 mm. of mercury and 9.7 mm. of mercury, respectively, lower one hour after lying down than immediately after lying down.

4. The systolic and the diastolic blood pressure were 3.5 mm. and 2.8 mm., respectively, lower one hour after lying down than in the original sitting position.

5. The declining curves for the systolic and diastolic pressures were parallel.

6. The higher the increase in the blood pressure was immediately after lying down, the steeper was the declining curve during the first stage.

7. It is Kamogawa's opinion that the decrease in the blood pressure after lying down is due to local regulation, especially in the area of the retinal artery and in the blood vessels subject to the splanchnic nerve; the natural decrease in the action of the heart during reclining is contributary.

8. No distinct change in the pulse pressure was observed in the retinal artery.

9. No change in the general blood pressure or in the frequency of the pulse rate was noted.

K. L. STOLL.

Refraction and Accommodation

REFRACTIVE ERRORS AND COLOR OF THE IRIS. R. BASSIN and B. ŠKERLJ, Klin. Monatsbl. f. Augenh. **98:** 314 (March) 1937.

Bassin and Škerlj examined the relation of refractive errors, follicular conjunctivitis and blepharitis to the color of the iris in 2,848 boys and 1,964 girls. The ages ranged between 6 and 22 years. The color was recorded according to the charts of Martin; the refraction was determined with the skiascope, and no mydriatics were used. The results of these examinations were as follows: 1. Myopic and hyperopic errors of a medium or a high degree were more frequent in light-colored eyes (light gray and blue) than in those of a darker hue (brown, hazel and grayish hazel). 2. Refractive errors of a slight degree, i. e., myopia up to 2 D. and hyperopia up to 1 D., predominated in darker eyes. 3. Follicular conjunctivitis and blepharitis occurred twice as often in blue and gray eyes as in light gray eyes and about one third more often in brown eyes than in light gray eyes. 4. The findings were evenly distributed in both sexes. 5. Lack of pigment may be a sign of general biologic inferiority, and racial differences may influence the results obtained.

K. L. STOLL.

Retina and Optic Nerve

THE PROGNOSIS IN PAPILLOEDEMA. G. HOLMES, Brit. J. Ophth. **21:** 337 (July) 1937.

In this article only that form of papilledema associated with increased intracranial pressure is considered. It is well known that even intense

papilledema may not cause any disturbance of vision, though it is not so generally recognized that vision may remain unimpaired for long periods, in other words, that the papilledema may not be progressive. In cases of this condition there are probably quiescent or slowly growing tumors, permitting adaptation, so that there is no great or sudden rise of pressure. In rarer instances the papilledema subsides without surgical intervention or other specific treatment, and vision remains unaffected. This shows that papilledema as such does not always demand surgical intervention. The more rapidly the congestion and swelling of the optic disk develops, the more intense does the papilledema become and the greater the danger to the sight if the intracranial pressure is not relieved. Swelling of 5 D. or more is usually a danger signal warning against undue delay. Great engorgement of the retinal veins and early and extensive hemorrhage on the surface of the swollen disk may be regarded as the most important measure of the severity of the process.

Contraction of the arteries may be regarded as the first evidence of constriction in the structures in the nerve head by the organizing fibrous and glial tissue, and irreparable damage of the nerve fibers soon follows. Holmes agrees with Paton that transient loss of vision is usually due to compression of the chiasm and optic tracts by the bulging or distended floor of the third ventricle. When this temporary amaurosis occurs in association with a highly swollen disk it is to be regarded as a threat of permanent failure of vision.

When visual acuity is seriously reduced before operation appreciable recovery rarely takes place. Vision may also fail permanently after a decompression operation in cases in which neither the changes in the fundus nor the visual fields suggested the risk of such a catastrophe.

The article should be read in the original.

W. ZENTMAYER.

ATROPHIC RECESSION OF THE LAMINA CRIBROSA. A. C. REID, Brit. J. Ophth. **21:** 361 (July) 1937.

A woman aged 70 years had obvious deep cupping and wide exposure of the lamina cribrosa associated with a decidedly subnormal intra-ocular tension. Vision was 6/6, and the fields were practically full.

There was no history of attacks suggesting raised tension.

Reid contends that this case represents a group in which needless and useless operations are today being done.

The essential thing to bear in mind is that such recession of the lamina cribrosa and even atrophy may occur independently of any rise in the intra-ocular pressure. Three conditions may be envisaged which may combine or, for a time, at least, remain separate, and all three may occur independently of increased intra-ocular tension: (*a*) primary atrophy of the nerve elements, (*b*) cavernous atrophy involving both the nerve and the supporting tissue and (*c*) atrophic recession of the lamina cribrosa. No conception of retraction is needed to explain (*c*). In this case the picture was a combined one of (*b*) and (*c*), i .e., atrophic recession of the lamina cribrosa associated with involvement of the nerve elements. The term "atrophic recession" seems adequate to explain the condition. Such recession is likely to be accompanied by atrophy, first

of the glial elements in front of the lamina cribrosa, thus leading to a wider exposure. To quote Elliot, the problem "is not the simple one of the physical effects of pressure alone." Hence may not the factor other than pressure alone be the blood supply of the glia (central artery) and of the lamina cribrosa (the circle of Haller)?

W. ZENTMAYER.

A CASE OF OGUCHI'S DISEASE IN SWEDEN. K. O. GRANSTRÖM, Klin. Monatsbl. f. Augenh. **98:** 77 (Jan.) 1937.

Only a few cases of Oguchi's disease have occurred outside of Japan. Granström records these and adds one case of his own observation. A man aged 25 showed yellowish white spots in the fundus, which appeared perfectly normal after one half hour of adaptation in the dark room. He complained of hemeralopia since childhood, and his sensitiveness to light was considerably reduced when tested with Gullstrand's photoptometer. The visual fields were normal for white, red and blue, and the perception of color was normal. The disks showed no abnormality. In the fundus of each eye a diffused slate-colored, or grayish-whitish yellow, discoloration was noted, which was most distinct in the periphery. The central retinal vessels, which were normal as a whole, seemed to be raised above the level of the retina in some places. Numerous reflexes were seen all over the fundus, including the macular region; the fovea appeared brighter red than usual, resembling its appearance in cases of embolism of the central retinal artery. The design of the choroidal vessels could be observed only after adaptation in the dark room, when the retinal phenomena had practically disappeared.

There was distant consanguinity of the parents. No other member of the family had hemeralopia; the patient's only sister was myopic, but her sensitivity to light was hardly more reduced than in many myopic persons.

The Wassermann reaction was negative, and the patient belonged to blood group O, blood type N. He was of the Nordic type, a mixture of the Svea and the Göta type, with possibly a slight Semitic admixture. Granström is convinced that his diagnosis of Oguchi's disease is correct.

K. L. STOLL.

LIPOID IN THE PIGMENT EPITHELIUM IN RETINITIS PIGMENTOSA: REPORT OF A CASE. R. ASAYAMA and A. TAKAGI, Klin. Monatsbl. f. Augenh. **98:** 162 (Feb.) 1937.

The right eye of a man aged 56 became blind as a result of retinitis pigmentosa and simple glaucoma. Atrophy of the optic nerve and edema of the choroid were seen histologically The retina, choroid and pigment epithelium showed the typical degeneration. In the degenerated portion and also in the pigment cells which had migrated into the retina granules of lipoid of varying size were observed. They differed distinctly from granules of fuscin and consisted of free oleic acid. This process inhibited the formation of visual purple, suggesting a relation of this disease

to hemeralopia. The authors concluded that diffused steatosis of the pigment epithelium is not a requisite for the occurrence of hemeralopia. Calcified drusen were present in the pigment epithelium, and some were deposited on the glassy membrane.

K. L. STOLL.

Trachoma

NEW EXPERIMENTAL RESEARCHES IN TRACHOMA. CUÉNOD and R. NATAF, Ann. d'ocul. **174:** 433 (July) 1937.

Cuénod and Nataf introduce the subject with a review of the work which they, in association with Charles Nicolle, have been carrying on It is over thirty years since one of them (Cuénod) with Nicolle began the experimental study of trachoma and arrived at certain conclusions, which have been confirmed by recent pathologic examinations. In 1909 and 1911 these authors, continuing their researches, reported on a series of experiments done on the chimpanzee and man and demonstrated that the chimpanzee offers perfect receptivity and that in this animal the lesions are of the same type and run the same course as in man. In the beginning, trachoma is accompanied by neither inflammatory phenomena nor hypersecretion. The best method to bring on infection in the monkey is by means of scarification, and without doubt trachoma may begin in man after the most superficial abrasion of the conjunctiva. Finally, trachoma in the chimpanzee can be communicated by inoculation during a long period of its course; it is contagious in man under the same conditions, and not only at the beginning of the disease, as was formerly believed.

In reporting this new work Nicolle and Cuénod definitely demonstrated the insidious character of the onset of trachoma and the lengthy period that it was contagious. To crown these experiments they demonstrated in 1912 that the agent of trachoma is a filtrable virus.

Cuénod and Nataf describe in detail their experimental work in their study of the virus and the details of the study of the trachomatous tissue and scrapings from the follicles of trachoma, in which they found what they call *plastilles* (drops of plasma) and which they believe to be analogous to the inclusions observed in the epithelial cells by Prowazek and Halberstädter.

At different times they inoculated lice, guarded against any outside contamination, with trachoma virus, and the result was always a veritable swarm of rickettsial elements in the intestines, while the control lice remained normal. On the other hand, in many cases of infantile folliculosis or of nontrachomatous conjunctivitis the inoculation of lice with conjunctival discharge always gave negative results. On the basis of the results of their inoculation experiments with monkeys the authors believe, first, that the trachoma virus may well be classed in the rickettsia family and that it may be more or less transmitted by agents of this family, and, second, that if lice are not the only transmitters of trachoma they may constitute more or less a reservoir for the trachoma virus.

The article is an extensive one and should be read in the original, as it is too long for a complete abstract.

S. H. MCKEE.

ETIOLOGY OF TRACHOMA. A. CUÉNOD and R. NATAF, Rev. internat. du trachome **14:** 117 (April) 1937.

The authors are convinced that the etiologic agent of trachoma is a rickettsia-like body. They propose the hypothesis that in families infested with lice the children infect their fingers by scratching, and then by rubbing their eyes transfer the infection.

J. E. LEBENSOHN.

Tumors

TUMOR OF THE CHOROID: METASTASIS IN THE LIVER EIGHT YEARS AFTER ENUCLEATION. P. DUPUY-DUTEMPS, Bull. Soc. d'opht. de Paris, May 1937, p. 284.

The patient was first seen in 1928 at the age of 33 years. Visual acuity of the left eye had gradually diminished to perception of fingers at 20 cm. The ophthalmoscope revealed a pigmented tumor of the choroid near the posterior pole with extensive detachment of the retina. The ocular tension was not raised. Enucleation was carried out, and histologic examination confirmed the diagnosis of nevocarcinoma. The patient has been watched carefully since that time. Eight years afterward, in 1936, hepatomegalia was discovered. Attending symptoms were fever, digestive disturbances and pain over the hepatic zone. Exploratory laparotomy was done, and a portion of the hepatic tumor was removed for biopsy. A photomicrograph of a section of the tumor of the eye and one of a section of the tumor of the liver are shown for comparison. Dupuy-Dutemps mentions also ten other cases of tumor of the choroid. In three of these enucleation had been performed sixteen, thirteen and nine years before, respectively. In two other cases no sign of metastasis had appeared after three and four years, respectively. The other five patients showed a metastatic growth in the liver after six months, one year, three years, five years and eight years, respectively. Metastasis in the liver is considered elective.

L. L. MAYER.

TUMOR OF THE OPTIC NERVE. S. BRAUN-VALLON, Bull. Soc. d'opht. de Paris, May 1937, p. 287.

A boy 3 years old was brought to the clinic because of recent deviation of the right eye. The first signs noted by the mother were intermittent deviation downward, together with slight protrusion of the globe. There was no history of trauma or infection. On examination marked deviation downward, with moderate exophthalmos, paralysis of the superior rectus muscle and limitation of motion on abduction and adduction, was noted. The exophthalmos was partially reducible, and there were no signs of inflammation. Ophthalmoscopic examination revealed slight papillary stasis and tortuous and dilated veins but no hemorrhages. The visual acuity of the affected eye was greatly diminished, but owing to the child's age the visual field could not be measured. The left eye was normal. General and neurologic examinations gave no positive findings. Roentgenograms showed failure of the cranial sutures to close and enlargement of the optic canals, especially that on the right.

At operation a tumor encircling the optic nerve, extending from just back of the globe to the optic canal, was found. It was necessary to remove the optic nerve with the tumor. The exophthalmos receded, but the deviation downward and the paralysis of the superior rectus muscle persisted. The histologic diagnosis was epithelioid meningoblastoma invading the nerve fibers. A picture of the child and a photomicrograph of a section of the tumor are included.

L. L. MAYER.

Uvea

DOYNE'S CHOROIDITIS. J. MALBRÁN and E. ADROGUÉ, Arch. de oftal. de Buenos Aires **11:** 529 (Sept.) 1936.

A case of Doyne's choroiditis which was not familial and was characterized by honeycombed, white retroretinal deposits in the macular region is reported. A review of similar cases reported by Hutchinson and Tay, Doyne, Mould, Helthouse and Batten, Bikerton, Treacher Collins Lloyd, Stargardt and Behr is given, and distinctive features are pointed out.

C. E. FINLAY.

PURULENT IRIDOCYCLITIS WITH GONOCOCCI WITHIN THE EYEBALL: REPORT OF A CASE. C. VELHAGEN SR., Klin. Monatsbl. f. Augenh. **98:** 20 (Jan.) 1937.

A girl aged 20 had slight congestion of the left eye, which changed to iritis, with deposits on Descemet's membrane, after eighteen days. A month later, a hypopyon developed, and a yellow reflex could be observed in the vitreous. In two weeks the lens became totally opaque, the hypopyon increased in size, perception of light failed, and the pain became intolerable, owing to the enormous intra-ocular tension. The latter symptom is unusual in cases of a purulent intra-ocular process. The eye was removed two months after the onset of the inflammation. Gonococci were observed in the lens, ciliary body and ora serrata, although repeated gynecologic examination proved their absence in the vagina, cervix and urethra. The glaucoma was caused by the tumescence of the lens brought on by the bacteria, which invaded the anterior chamber after rupture of the anterior capsule of the lens.

K. L. STOLL.

Therapeutics

LYSOTHERAPY IN DISEASES OF THE EYE. S. M. KHAYUTIN and E. I. ZLATKINA, Sovet. vestnik oftal. **6:**713, 1935.

The technic of preparing and of using the lysates is given. A preliminary study of the effect of lysates on the eye was made on a number of rabbits. One hundred patients with various diseases of the eye were treated with lysates. Most of these suffered from chronic diseases in which all other means of treatment were unsuccessful. Lysotherapy gave positive results in ten cases of keratoscleritis. A remarkable improvement was observed in twenty-two patients suffering from phlyc-

tenular keratoconjunctivitis; photophobia, epiphora and the phlyctenae disappeared after from two to four intramuscular injections of lysates. In three of seven patients who suffered from sympathetic ophthalmia, there was a marked improvement in the sympathizing eye. In nine of ten patients with plastic iritis, the pain and irritation of the eye decreased after the third or fourth injection. No favorable results were obtained in vitreous opacities, zonular cataract, chorioretinitis and deep keratitis.

Khayutin and Zlatkina came to these conclusions: 1. Lysotherapy is definitely favorable in scleritis, phlyctenular keratoconjunctivitis and iritis. 2. Lysates in small doses should be included in the treatment of sympathetic ophthalmia as a preventive against threatening blindness. 3. In severe diseases of the eye the dosage of lysates should be small. 4. Injections of lysates are painless and cause no general reaction.

O. SITCHEVSKA.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Clinical Meeting, London, England, Friday, Oct. 8, 1937

MR. W. H. MCMULLEN, O.B.E., F.R.C.S., *President*

A CASE OF DEFECTIVE MOVEMENT OF THE LEFT EYE. MAJOR J. BIGGAM, R.A.M.C.

A youth of 18 years who had been training at the Army technical school as a bricklayer was well until one night, when he felt ill, possibly because of something he had eaten. He vomited three or four times, and, in case it should be wanted, a bucket was placed at his bedside. This he struck in the night with his left eye, and a bruise resulted. In the morning when he awoke he felt still unwell and had diplopia. The latter, however, improved slightly for a month or so. All forms of investigation gave a completely negative result, and the Wassermann reaction and the results of neurologic investigation were negative. As there was liability of a fall from a scaffold and injury of himself or other persons, he was "invalided out." In reading a book he tilted his head backward. He is now in full civilian work. He has binocular vision inside a certain area. The opinion of Dr. Gordon Holmes was asked, and Dr. Holmes wrote that the fact that upward and downward movements were involved, rather than movements of individual muscles, suggested that the patient had a supranuclear lesion, and advised carrying out the Roth-Bielschowsky test. This test is considered positive if the eye moves from the position of rest when it is fixed on any object, and the head is moved in the opposite direction. The patient is asked to look at one's face and then slowly flex his head downward. If the lesion is supranuclear, the left eye should move outward.

DISCUSSION

MR. L. H. SAVIN: In the London County Council hospitals last year there were a number of cases of encephalitis which were supposed to be related to the epidemic of influenza.

LENTICONUS POSTICUS. MR. T. HARRISON BUTLER.

Lenticonus posticus has been confused with myopic degeneration of the lens. The latter is due to the fact that the lens is composed of a concentroid mass of nuclei which have a gradually increasing curvature. There may be senile sclerosis of the center of the lens without association with any alteration of the index of refraction, and there is no increase or development of myopia in cases in which that had been nonexistent. Now and then, however, one meets with a case of this

condition associated with a changed refractive index. If some parts of the lens have a higher index of refraction, rays passing through such parts will come to a focus closer to the lens than those which pass through the periphery, resulting in a linear focus. Sometimes one is puzzled by persons declaring that everything they look at appears watery, yet examination by ordinary methods reveals nothing amiss. In such cases, though vision may be 6/4 as examined by test types, some retrobulbar disorder should be suspected. Or the condition in the lens may be one of those which can sometimes be elucidated by the use of the slit lamp. In these cases the myopia may increase with extraordinary rapidity.

Lenticonus posticus is an uncommon condition; perhaps one person in ten thousand has it. On looking into the lens with the slit lamp one sees what looks like a globule of oil, and on flashing the light round one makes out a crater-like appearance. One may come across the condition known as lenticonus posticus internus. I show views illustrating this disorder in two patients, sisters. It was said that there was a family history of the same condition.

A condition which it is necessary to distinguish the foregoing from is glass blowers' or puddlers' cataract. The illustrations I present show peeling off of the zonular lamella of the anterior cataract. The slit lamp shows at once what has happened. The zonular lamella is the outermost part of the capsule, and when the lens is dislocated by violence it is dislocated out of the zonular lamella. The latter remains behind, with the filaments of the suspensory ligament attached to it. When one finds a posterior central opacity combined with peeling off of the zonular lamella, one can almost always say with certainty that it is the result of infra-red irradiation. But I do not think that one can go into a court of law and state that because the patient is a glass blower or a puddler he is therefore suffering from puddlers' cataract; one can say no more than that it is almost certainly so.

I also show pictures of radium cataract. This may result a long time—as much as two years—after the applications of radium. The slit lamp shows large vacuoles just under the capsule. One can see a rarefied space, and definite secondary lenticonus posticus.

(A number of photographs and drawings are demonstrated.)

DISCUSSION

MR. J. FOSTER: I have seen cases of glass blowers' cataract in persons working at the making of glass bottles. One man with this condition had been working on red-hot blooms with a steam-hammer for twenty years. He had only one sound eye, and it showed desquamation of the capsule, a curling up of the lamellar layer. A peculiarity of the law is that in cases of this injury compensation can be paid only if the operation had been done within six months after the accident.

DR. A. J. BALLANTYNE, Glasgow: Referring to the changes due to myopia, I had as a patient a woman who had been wearing a —13 D. sphere because of myopia associated with condensation of the nucleus of the lens. She was so discontented with her lens that she had extraction carried out by another physician, but as she then had to wear a + 13 D. sphere the contrast was so great that she never became reconciled to it.

MR. BASIL GRAVES: Optical section of the type of sclerosed nucleus referred to by Mr. Harrison Butler reveals a feature which gives added interest in conjunction with Fincham's recent monograph (The Function of the Lens Capsule in the Accommodation of the Eye, abstr., *Brit. J. Ophth.* **19:** 51, 1935). The article points out certain characteristics relating to the shape, in accommodation, of the anterior capsule, the curvature of which is complicated by shortening of the radius of the central portion. The anterior face shows the same type of curvature, while the posterior face has a relatively uniform curvature. This provides an interesting subject for speculation.

Book Reviews

Medical Treatment of Cataract. By A. Edward Davis, M.D. Price, $3. Pp. 165, with 11 illustrations. Philadelphia: F. A. Davis Company, 1937.

A. Edward Davis, formerly professor of ophthalmology and consultant in ophthalmology of the New York Post-Graduate Medical School and Hospital (Columbia University), has given in a book of one hundred and sixty-one pages an elaboration of the medical treatment of cataract. His object has been to learn the cause of senile cataract and a method of retarding its progress or of clearing up the opacities. His efforts in this field have been persistent over a period of years. The book is divided into twelve chapters. The historical aspects of the subjects are given, followed by a review of the various theories of the etiology of cataract. The importance of early diagnosis is stressed; the pupil should be dilated to afford complete examination of the crystalline lens. If incipient cataract is found, the patient should have the condition explained to him so that he may more readily agree to the necessity of an investigation of his general condition and cooperate in the treatment which is indicated.

The symptoms and signs of cataract are detailed in chapter 4. Under the pathogenesis of cataract, the permeability of the capsule, the effect of toxins, specific and otherwise, calcium and other substances and endocrine deficiencies are considered. A chapter is devoted to spectroscopy of the lens. The greater portion of the book is devoted to the discussion of the treatment of cataract. The effect of correction of errors of refraction is given by J. Burdon Cooper. The dietary treatment is elaborated on, and the use of ethylmorphine hydrochloride, mercury cyanide, iodides, vitamins, glycerin, boric acid and other agents is discussed.

The book, however, seems to be devoted to the defense of the treatment of cataract by the injection of lens antigen as previously advocated by the author in various papers. To establish the rationale of the method Davis quotes: (1) J. Burdon-Cooper ("Subcapsular senile cataract is undoubtedly a chemical decomposition of the lens proteid"), (2) Roemer ("It is a specific metabolic disease of the lens" and "due to a direct toxic action upon the cells and fibers of the lens") and (3) Carrel ("Certain blood cells, e. g., lymphocytes, have a double function, destroying foreign substances and necrotic material and promoting cell proliferation").

Davis himself states: "Lowered nutrition, general and local, together with toxins in the blood, must be held responsible for most senile cataracts." The injection of lens antigen causes the formation of specific antibodies and creates mild leukocytosis which fortifies the healthy fibers of the lens against toxins, while at the same time it destroys or liquefies the dead fibers of the lens and cataractous or necrotic material.

Are these premises acceptable for this thesis? The reviewer grants that specific antibodies may be formed by the injection of lens proteins

and that they are specific for the normal transparent lens proteins, but, not being specific for coagulated or necrotic lens protein, they cannot combine with the latter for its absorption. He thinks that they probably do not reach the lens, protected as it is by the semipermeable membranes of the ciliary epithelium and of the capsule. The normal aqueous contains little protein and a very low percentage of the antibodies which are present in the blood, but if the antibodies do enter the lens, the reaction would probably be precipitation of some of the remaining transparent lens protein, and this might add to the opacity. The leukocytosis created by injections of lens protein is hardly necessary or desirable in cases of uncomplicated senile cataract. Increased formation of lenticular fibers is not likely to result, as the growth of the lens is at a minimum in the age group of persons with senile cataract. Cataract has never been considered to be the result of, or accompanied by, inflammatory processes of the eye. In cases of uncomplicated senile subcapsular or cortical cataract, floating cells and an increased protein content of the aqueous are not demonstrable. The contention that the blood cells are stimulated to salutary action by injections of lens protein in cases of cataract is not proved.

Davis would apply his treatment only in cases of subcapsular cataract in which vision is better than 20/70. It is not suitable in these cases or in the great number of cases of nuclear or other types of senile cataracts. The book should contain a classification of cataract and state the relative incidence of the varieties. It should define accurately the subcapsular type which is suitable for treatment. The objective changes which occur in the crystalline lens in the development of cataract should be accurately recorded with the aid of the biomicroscope and photography of the lens, so that these can be used as criteria for the improvement which is noted in the cases reported. Well recorded observations of the clearing of vacuoles or globules, water-split sutures or separated lamellae or of the diffuse haze, features which constitute the development of the incipient stage of senile cortical cataract, would be well worth while. Satisfactory objective criteria must be decided and agreed on for the judgment of the clinical effects of the treatment of cataract. A statement of improvement in vision alone is not sufficient.

Davis' statement in the preface "If such knowledge were carried to the general practitioner and if early active treatment were established by the specialist, at least one-half (perhaps, three-fourths) of all operations for senile cataracts as now performed, would be rendered unnecessary," is certainly unfair and misleading to the public. The advertisements by the publisher are particularly misleading and should not be permitted, because the statements contained therein have not been proved. Simply on the basis of the incidence of subcapsular cataract in relation to that of nuclear and other forms of cataract, which are admitted by the author as being not amenable to his treatment, the statement is disproved. Dr. Davis' work is an effort to eradicate one of the tragedies of human life and in this respect is to be commended, but his thesis on the arrest and cure of senile subcapsular cataract by injections of lens antigen may not be accepted in book form any more than it has been in his published papers. Perhaps the reason why his work has not been accepted is that he has not set forth the objective data in regard to his cases in a scientific manner. The field of the investigation of cataract, with

reference to its cause, prevention and cure is an important one. Any treatment carried on in a scientific manner is a legitimate experiment. The evaluation of clinical results in this work is difficult, and all available objective criteria should be used. It is to be hoped that in any future reports these demands will be satisfied.

DANIEL B. KIRBY.

Ueber Retinitis unbekannten Ursprungs nebst einer histopathologischen Studie über Misbildungen des Sehorgans. By Carl Heijl, Stockholm. Price, 18 marks. Pp. 40, with 108 illustrations on 50 plates. Jena: Gustav Fischer, 1937.

There is a group of cases of chorioretinitis in which the cause remains undetermined; the condition has been regarded as either inflammatory or degenerative, on the one hand, or due to constitutional causes, on the other.

Formerly most conditions of the eye, including coloboma, microphthalmus and other developmental defects, were regarded as inflammatory. Recently interest in heredity and developmental disturbances has been aroused, and many conditions have been found to be inherited. These changes in the eye follow the same laws as changes elsewhere. Nevus and birthmarks are disseminated deformities of tissue, localized in the mucous membranes, meninges, central nervous system and eyes. Heijl discusses the relation of tuberous sclerosis and neurofibromatosis to the formation of nevus in a most interesting way. The principal topics of this book, however, are retinitis exudativa externa, or Coats' disease; congenital retinal detachment; pseudoglioma (the formation of connective tissue posterior to the lens and persistent hyaloid artery); microphthalmus; orbitopalpebral cysts, and other deformities.

These conditions were always regarded as the result of intra-uterine inflammation, but the author, after his study of ocular rudiments in teratomas, came to the conclusion that there is an unusual analogy between these conditions and the conditions under discussion. He had access to the ophthalmic and pathologic specimens in Stockholm, Upsala and Lund, and the investigation was carried out in Professor Henschen's Pathological Laboratory in Stockholm. The material examined was that in two cases of Coats' retinitis, eight cases of congenital retinal detachment, one case of formation of connective tissue posterior to the lens, one case of microphthalmus associated with coloboma of the iris and two cases of orbital cysts. Heijl also discusses gliosis, rosette formations, pigment cells, anomalous blood vessels and intra-ocular defects of mesodermal origin.

The author concludes that while Coats' disease and congenital retinal detachment were formerly regarded as examples of "ophthalmias," this interpretation is not convincing. A study of problems of heredity and developmental errors has shown that the eye, on account of its complicated structure, is frequently affected by developmental defects and other constitutional infirmities.

In studying this question the author has followed a new scheme, namely, a comparison of the histologic features of pseudoglioma and of severe developmental disturbances which are present in microphthalmus, synophthalmus, orbital cysts and the eyes of teratomas. This has shown

that in all these conditions there are a number of pathologic peculiarities present which can only be defects in development. He concludes that most conditions known as pseudoglioma are developmental defects.

Another question of great importance concerns the nature of the inflammatory and degenerative processes which were present in these cases; the study of this material convinced the author that the degenerative and inflammatory processes which formerly were regarded as the cause are really secondary and result from poor reactive powers of deformed tissue and from its reduced power of resistance. The author also endeavors to explain the occurrence of developmental defects in the glia, the neural epithelium and pigment epithelium.

The book is illustrated with 108 photomicrographs on 50 plates, in which the pathologic changes in these conditions and in other developmental defects are shown.

Heijl has written an excellent and thoughtful treatise on a difficult subject, which suggests a new explanation of important conditions and should stimulate further investigation.

ARNOLD KNAPP.

Diseases of the Nose and Throat. By Sir St. Clair Thomson, M.D., F.R.C.P. (London), F.R.C.S., LL.D. (Winnipeg), Médecin Diplome en Suisse, Emeritus Professor of King's College Hospital and the Royal Ear Hospital, London, and V. E. Negus, M.S. (London), F.R.C.S., Surgeon for Diseases of the Nose, Throat and Ear, King's College Hospital, London. Fourth edition. Price, $14. Pp. 976, with 402 illustrations. New York: D. Appleton-Century Company, Inc., 1937.

Since the publication of the first edition twenty-five years ago, this work has been regarded as one of the most acceptable on the subject of rhinolaryngology. The fourth edition now appears, thoroughly revised yet maintaining the original plan of Thomson.

This is a book of personal experience, written in a delightful style, offering a valuable and safe guide for the physician engaged in this specialty as well as constituting a satisfactory work of reference. The text is in large and small type in accordance with the importance of the subject. The numerous bibliographic references are to be found as footnotes at the bottom of each page instead of at the end of the chapter—a method which has much in its favor.

The chapters on sinus disease and the larynx are fully presented. The part on peroral endoscopy has been entirely rewritten. Neoplastic growths everywhere in the upper respiratory tract have been accorded a prominent place, with especial reference to removal both by surgical intervention and by diathermy. The subject of syphilis occupies less space than in former editions, as does also that of diphtheria and that of the once important method of laryngeal intubation. More exact means of diagnosis and treatment have brought about these changes. The elusive subject of allergy has not been discussed in much detail, perhaps because the English mind has not yet accepted the protean manifestations of this disease which have been admitted on this side of the Atlantic.

Of especial interest to the ophthalmologist is the part on the treatment of the various foci of infection, those circumscribed regions in which pathologic microorganisms proliferate, giving rise to systemic or local disturbances of serious import.

A septic focus in a tonsil may be suspected when a creamy drop or two of pus can be expressed from the fossa, when the tonsil has a congested sodden appearance and likewise when there is a reddish color of the injected anterior pillar, often accompanied by enlargement of the cervical glands.

Bacteriologic examination cannot be relied on, for one can often procure hemolytic streptococci in pure culture from material from a person in absolute health. The results of tonsillectomy for secondary infection are therefore uncertain.

According to the authors, in less than 4 per cent of the cases of retrobulbar optic neuritis is the condition found to be caused by sinusitis. Many patients recover spontaneously, or the condition may occur as an early symptom of multiple sclerosis. However, if one naris is stuffy and pus can be seen above a red or swollen middle turbinate, it is justifiable to uncap the ethmosphenoid cells.

Attention is drawn to the fact that in the operation for mucocele the bony walls are often eroded, with exposure of the dura. A drainage tube should be introduced down through the nose and retained in situ for a week or more after removal of the cyst.

All malignant growths in the sinuses and orbit should be treated surgically as far as possible, and removal should be followed by irradiation.

To review a book of this character is indeed a pleasure.

HAMPTON P. HOWELL.

The Ocular Fundus in Diagnosis and Treatment. By Donald T. Atkinson, M.D., F.A.C.S. Cloth. Price, $10, net. Pp. 259, with 106 engravings, including 58 colored plates. Philadelphia: Lea & Febiger, 1937.

In this book, which is intended for the use of surgeons, obstetricians, neurologists and other than ophthalmologists, Atkinson has endeavored to "outline the general characteristics of the ocular fundus, in health and disease." As he believes that "realism and accuracy are likely to be enhanced in illustrations done by the ophthalmologist, who himself studies the details of pathology in each case," the book becomes the only one of its kind in English in which the author has himself made the illustrations.

The text consists of eight chapters, dealing in turn with the use of the ophthalmoscope, the normal fundus, pathologic changes in the retinal vessels, the optic nerve, the vitreous, the retina, the choroid and, finally, ophthalmoscopic changes in special diseases. These chapters contain forty-eight schematized drawings in black and white. In connection with most of the various pathologic entities there is a brief section devoted to treatment. These sections serve only to make ambiguous the title of the book. They might well have been omitted.

The colored plates are fifty-eight in number, and it is presumably by these that the usefulness of the book is to be judged. When it is

remembered that the book is not intended primarily for the use of ophthalmologists, it would seem that some objection might be raised in regard to the lesions selected for illustration. Of the fifty-eight plates, eight represent normal fundi. Of the remaining fifty plates, more than thirty illustrate cases interesting enough to have been referred to the author. The chief difficulty which besets the physician who makes only incidental use of the ophthalmoscope is not the recognition of choroidal leprosy, a dipterous larva in the vitreous, Cysticercus cellulosae or even angioid streaks. His great problem is to recognize a normal fundus or to recognize and appraise slight departures from the normal. In this respect Dr. Atkinson's pictures will afford little help, for the conditions portrayed, when not of inherent rarity, become unusual because of extreme severity or extent.

Finally, and perhaps most important of all, Dr. Atkinson has failed to impart a sense of realism, as far as the reviewer is concerned. On the contrary, many of the plates give rise to a decided impression of artificiality. The hemorrhages and exudates depicted seem, almost without exception, much too obvious. On the other hand, nowhere do attenuated arteries seem sufficiently attentuated. The colors of the atrophic nerve heads, especially of those showing glaucomatous atrophy, are unusual, while the picture of the coloboma of the optic nerve comes perilously close to being unrecognizable. The author has undoubtedly expended much time and effort to produce this book. The reviewer believes it fair to say, however, that the field covered has been covered before and covered much better.

O. P. PERKINS.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.
President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the President, Dr. P. Bailliart.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Costzeedijk 316 Rotterdam, Netherlands.
Place: Cairo. Time: Dec. 8-14, 1937.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. P. E. H. Adams, 6 Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13 Mitchell Ave., Jesmond, Newcastle-on-Tyne.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.

Midland Ophthalmological Society

President: Dr. T. E. Ashdown Carr, 34 Charnwood St., Derby, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.
Time: Oct. 1, 1937.

Ophthalmological Society of Egypt

President: Prof. Dr. Sayed Abdel Hamid Soliman Pasha, Faculty of Medicine, Cairo.
Secretary: Dr. Abdel Fattah El Tobgy, 3 Midan Soliman Pasha, Cairo.
Time: March 1938.

Ophthalmological Society of the United Kingdom

President: Mr. Gordon M. Holmes, 9 Wimpole St., London, W. 1.
Secretary: Mr. J. D. M. Cardell, 27 Weymouth St., London, W. 1.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 7-9, 1938.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arieh Feigenbaum, Abyssinian Str. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel-Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warszawa.
Place: Lindley'a 4, Warszawa.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Ransom Pickard, 31 East Southernhay, Exeter, England.
Secretary: Dr. A. Rugg-Gunn, 35 Harley St., London, W. 1.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris, 7è.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

TEL-AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Allenby Str., Tel-Aviv.
Secretary: Dr. Sadger Max, 9 Bialik Str., Tel-Aviv.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: San Francisco. Time: June 13-17, 1938.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Frederick H. Verhoeff, 243 Charles St., Boston.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: San Francisco. Time: June 9-11, 1938.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.

SECTIONAL

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Rd., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. William D. Donoher, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: Victoria, B. C. Time: June 21-24, 1938.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. W. Howe, 740 St. Helens Ave., Tacoma, Wash.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Frank W. Broderick, 501 Central Trust Bldg., Sterling, Ill.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501—7th St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. Andre Cortopassi, 703 Second National Bank Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. H. Judd, 107 S. 17th St., Omaha, Neb.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.
Place: New Orleans. Time: Nov. 30-Dec. 1-3, 1937.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. John Hunter McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. Wearne Beals, Weber Bldg., DuBois.
Secretary-Treasurer: Dr. C. Wearne Beals, Weber Bldg., DuBois.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Charles T. Flynn, 41 Trumbull St., New Haven.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Place: New London. Time: February 1938.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. John King, Thomasville, Ga.
Secretary-Treasurer: Dr. Mason Baird, 384 Peachtree St., Atlanta, Ga.
Place: Augusta. Time: May 1938.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. E. E. Holland, 51 S. 8th St., Richmond.
Secretary: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: April 6, 1938.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. H. Lamb, American Bank Bldg., Davenport.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.
Place: Davenport.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. C. A. McWilliams, G. and S. I. R. R. Bldg., Gulfport, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Place: New Orleans. Time: Spring, 1938.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. F. N. Smith, Grand Rapids Clinic, Grand Rapids.
Secretary: Dr. Dewey R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Oram Kline, 414 Cooper St., Camden.
Secretary: Dr. James S. Shipman, 542 Cooper St., Camden.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Marvin F. Jones, 121 E. 60th St., New York.
Secretary: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Time: May 10, 1938.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. Rosenberger, 221—5th St., Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, 514—6th St., Valley City.
Place: Bismarck. Time: May 1938.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George Truluck, Orangeburg.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Kate Savage Zerfoss, 165—8th Ave. N., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 805 Medical Arts Bldg., Memphis.
Place: Nashville. Time: April 12-13, 1938.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
Place: Fort Worth. Time: Dec. 11 and 12, 1937.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. M. H. Hood, 505 Washington St., Portsmouth.
Secretary-Treasurer: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. James A. Fisher, 501 Grand Ave., Asbury Park, N. J.
Secretary: Dr. William F. McKim, 488 Sanford Ave., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Andersen, 106 S. Main St., Akron.
Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. William C. Warren Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Henry F. Graff, 513 N. Charles St., Baltimore.
Secretary: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Thomas D. Allen, 122 S. Michigan Blvd., Chicago.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Webb Chamberlin, 7405 Detroit Ave., Cleveland.
Secretary: Dr. Walter H. Rieger, Hanna Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. Paul Moore, Republic Bldg., Cleveland.
Secretary: Dr. G. Leslie Miller, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. C. E. Silbernagle, 247 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. D. M. Johnson, 327 E. State St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. Edgar G. Mathis, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Lester H. Quinn, 4105 Live Oak, Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. J. D. Carroll, 102—3rd St., Troy, N. Y.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. L. Allen, 1215 Walker Ave., Houston, Texas
Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. C. P. Clark, 24 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. O. S. Gilliland, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. F. C. Hertzog, 117 E. 8th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. Isaac H. Jones, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Earle Breeding, 1801 I St., N. W., Washington.
Secretary: Dr. Elmer Shepherd, 1606–20th St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Thomas A. Judge, 735 N. Water St., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 208 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. A. G. Farmer, 1040 Fidelity Bldg., Dayton, O.
Secretary-Treasurer: Dr. Rome M. Webster, 663 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.
Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Guy Maness, Nashville, Tenn.
Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Jesse Stark, 45 Park Ave., New York.
Place: Squibb Hall, 745 5th Ave. Time: 7 p. m., first Monday of each month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. A. J. Young, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

OPHTHALMOLOGICAL SOCIETY OF THE UNIVERSITY OF PITTSBURGH

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, 351—5th Ave., Pittsburgh.
Time: Second Monday in November, January, March and May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. John S. Van Winkle, 297 Broadway, Paterson, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, 351—5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. A. F. Bagby, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Leslie Charles Drews, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Joseph W. Crawford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Raymond A. Lower, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Gordon D. Hoople, 110 Medical Arts Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W., Toronto, Canada.
Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 Eye St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.

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